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THE THIRD STAGE OF LABOUR

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Many doctors have told me that they have adopted practices which exclude midwifery mainly because of the third stage of labour. There can be but few—if any—doctors who have not been confronted with the frightening complication of this stage of labour which may act like a paralysing drug to the newly initiated. The impact may be so sudden and so severe upon the doctor that the decision to steer clear of it for the rest of his or her days is readily understood. This is the stage of labour following the birth of the baby. The mother feels that she has done her job and often is exhausted. The doctor and nurse may inadvertently relax in their trains of thought and action whereas they are witnessing and conducting a physiological act which in the flash of an eye may become so pathological as to cause the death of the mother. The management of this stage requires patience and courage, that is, masterly inactivity most times, yet rapid determined action when warranted, for the onset of severe complications is usually unexpected, sudden, and may be catastrophic. Should the patient survive a severe degree of collapse, she is still confronted by many endocrinal changes that may follow in the wake of shock. Obviously, this places a heavy responsibility on the accoucheur. It is self-evident that he should at all times have the basic principles of the physiology and management of this stage of labour and its complications at his fingertips—literally and figuratively.

The Placental Stage of Labour

During the first stage of labour we are taught that the uterine muscle not only contracts but retracts. This property is responsible for cervical dilatation. Once the cervix is fully dilated, further contractions together with maternal, and, possibly, assisted efforts, result in the delivery of the baby. The patient now is said to be in the third or placental stage of labour. Let us therefore examine what has happened to the placenta during the first 2 stages of labour since this may have a direct bearing upon the mechanism of the third stage, i.e. of placental loosening and expulsion. The view is held that when contraction and retraction occur in the uterine muscle, the region of uterine wall over the placental area, although affected, is not as markedly involved as is the rest of that organ's wall. This may be regarded as a life-saving mechanism since nature would not allow the vital supplies to her young in the very act of birth to be dangerously hampered. However, what happens in the wall of the uterus might not necessarily affect its contents to the degree imagined by us. What we do know, however, is that with the birth of the baby the uterine cavity shrinks and the uterine wall thickens at a comparative pace. This phenomenon may, often does, take a little while, i.e. the uterus does not necessarily change gears, it rests in neutral for a while. In lower animals, e.g. the pluriparous group, the young are expelled with amnion and placenta intact, i.e. there is a combination of the second and third stages. In the human, experimental work has been of great interest. Two studies are worth quoting. The first is Miss Burton Brown's work in Oxford. She injected radio-opaque material into the cord immediately after delivery, followed by taking serial X-ray photographs of the pertinent region. An immediate bunching up of the cotyledons reducing the placenta to half its size without separation was demonstrated. Once this had taken place, placental separation occurred in the presence of contraction and relaxation. Relaxation is necessary for the passage of the placenta into the lower segment. The average time taken for placental separation, as observed on utilizing this method, was 4½ minutes. In Liverpool they went one step further. Starting off with anencephalics, working their way through breech deliveries and eventually to vertex deliveries, radio-opaque material was injected into the cords of undelivered babies. Serial X-ray photographs were then taken. It was shown that as the foetus was being born, the uterine cavity contracted in a cylindrical fashion. Placental separation occurred very rapidly, i.e. almost at the time of foetal delivery. Both at Caesarean Section (during which time observation may be accurate but interpretation may be fogged because we are not sure whether an incised uterus, possibly in an anaesthetized patient, acts normally) and clinically (with a hand gently placed upon the uterine fundus in a thin patient one can feel the uterus working, altering its shape and expelling the placenta) the observed time taken for the placenta to separate varies tremendously from patient to patient.

The conduct of the third stage depends upon this clinical variation, i.e. the time taken for the placenta to separate and be expelled from the uterine cavity. The act of separation is to be completed before expulsion can take place. The signs of separation are therefore to be awaited. They are:

1. Lengthening of the cord.
2. A slight gush of blood.
3. The rather flat-topped, triangular-shaped uterus becomes hard, globular and mobile as it rises in the abdominal cavity.)
4. Slight suprapubic pressure causes further lengthening

of the cord. (Recession at this stage indicated that the acts of separation and expulsion from the uterine cavity have not been completed.)

Once the placenta has been expelled from the uterine cavity, contraction of the muscle fibres closes off the sinuses, thereby assuring no drastic haemorrhage. Maternal bearing-down efforts at this stage of the proceedings will result in the birth of the placenta, which slides out (Matthews Duncan) or peels out like an umbrella turning inside out (Schulze).

The management of the third stage, as has been stated before, requires patience and courage. The courage has to be twofold, *viz.* the more difficult one of being able to do nothing, in the normal case, and the easier but more dreaded one of having to act in a well-planned determined fashion when the pathological occasion arises. Therefore, in the normal course of events, immediately after birth, or when the cord has stopped pulsating, it is tied and cut. The baby is removed. The vulva is inspected. Now comes the problem —should the fundus be controlled or not, i.e. should one belong to the 'hands on' or 'hands off' school. As is usual in all vexed questions, it is the person with the reasonable outlook on matters who tends to get a grip on the issue. Should the patient be well-built, but relatively thin, the fundus can readily be seen as a well-defined abdominal lump. *Visual observation* is thus all that is required. Any variation from the normal, i.e., enlargement or disappearance of this lump is easily recognized. On the other hand, should size and obesity not allow visual observation, a hand may be placed gently on the patient's abdomen. The fundus is palpated and from then onwards *manual observation* is carried out. It is of paramount importance to be aware of what is happening to the uterus. Should it become flabby and distended with blood, early recognition will lead to prompt action and possible avoidance of unnecessary further complications. It must be stressed that should a hand be laid on the patient's abdomen, it is there strictly for purposes of observation. At no time should it be used as an adjunct trying to hasten physiological placental separation by stimulating undue uterine action.

Should there be no bleeding and the vulva requires stitching, this may be readily undertaken at this juncture. The stitches may be inserted but should not be tied until after the passage of the placenta. Premature narrowing of the gaping vulva may lead to unnecessary difficulties, e.g. the placenta may tear out the stitches in passage, or, should manual removal of the placenta be required, the stitches will have to be severed. Once there is evidence of placental separation, that organ is expelled either by the patient or the accoucher. Often a bearing-down effort is all that is necessary. Slight suprapubic pressure, together with applying tension to the cord, is a most satisfactory procedure for its removal. As soon as the placenta is born, it is examined to note whether it is complete or not and whether its membranes are more or less complete. Any abnormality is observed and noted. In the normal course of events the act of labour now is over. The baby is a helpless little individual requiring nurturing in order to carve out its own future. The patient has become a mother. Within her, certain physiological changes are rapidly taking place since milk has to be supplied whilst her pelvic organs return to normal.

DANGERS OF THE THIRD STAGE

What then are the dangers that have dogged the whole length of this stage and may even follow soon after it? The greatest amongst them are *haemorrhage* and *shock* (as a result of haemorrhage or trauma). Of nuisance value and forever threatening with the above complications, is retention of either the whole or portion of the placenta.

Haemorrhage

The haemorrhage that follows in the wake of the birth of the baby or placenta may be due to:

1. *Patent placental site sinuses*, which in turn are:

(A) Caused by poor contraction and retraction, due to (1) exhaustion, (2) poor management of the second stage (anaesthesia, analgesia, manipulation), (3) overdistension (e.g. multiparity, hydramnios) and (4) fibrous tissue—grande multipara;

(B) Kept open by: (1) partial separation of the placenta, (2) clot, (3) rigid walls: ? essential hypertension, and (4) concertina effect of uterus, i.e. if the uterus is down in the pelvis the arteries pump whilst the veins are blocked.

2. *Clotting defects:*
   (*a*) Some individuals bruise and bleed readily.
   (*b*) Afribrinogenaemia.
   (*c*) Blood dyscrasia.
3. *Trauma* of the cervix, vagina, vulva.

It is ever to be borne in mind that post-partum haemorrhage most often occurs when least expected. In 26,139 deliveries over 4 years in our institutions, post-partum haemorrhage

TABLE I. NUMBER OF CASES AND CAUSES OF POST-PARTUM HAEMORRHAGE

Unknown	1,451	63·4%
Known	752	39·9%
Clotting deficiency ..	8	0·4%
Traumatic	78	3·3%

occurred when least expected and without any apparent cause in 1,451 cases (Tables I and II). At all times must

TABLE II. HOSPITAL DELIVERIES, 1953-1956

Deliveries	26,139
Post-partum haemorrhage	2,289
Incidence of post-partum haemorrhage	8·7%

there be an awareness of this fact and whosoever is in charge of the delivery must be attuned to it mentally and physically.

It is difficult to define post-partum haemorrhage adequately. Individuals vary so tremendously in their reactions to blood loss. Of great importance in this reaction, amongst many others, is the haemic state before the onset of labour. The constant slow trickle is of as great importance as is the rapid gush of blood. For this reason it is both more scientific and accurate to measure the amount of blood lost by the patient. For statistical purposes a patient is classified as having suffered from post-partum haemorrhage if she had lost 20 fl. oz. of blood or more.

Treatment of Postpartum Haemorrhage

1. Uterine contraction has to be stimulated in order to close the sinuses. Uterine contents are to be expelled or removed, *viz.*

(*a*) Rub up the uterus.

(*b*) Express the placenta by Crede's method—or, should the placenta be out—express clot.

(*c*) Failure of Crede's method is to be followed by manual removal of the placenta.

(*d*) At all times must the expressed or removed placenta be inspected to make sure that it is complete.

(*e*) Keep the uterus contracted by the administration of oxytocic drugs e.g. ergot, pitocin.

Rarely is a uterus encountered that contracts but relaxes afterwards most readily, leading to further haemorrhage. Pitocin drip is of immense value in these cases.

2. Should the uterus be contracted, but be situated in the pelvis, it must be elevated into the abdomen in order to straighten out the concertina effect of the lower segment. Blood may then be returned to the systemic circulation by veins now rendered patent. These measures will obviate the use of bimanual compression, hot intra-uterine douches and packing.

3. If there is any interference with clotting, the appropriate constituents should be administered, e.g. fresh blood and fibrinogen, when indicated.

4. Lost blood should be replaced by adequate transfusion.

PREVENTION OF COMPLICATIONS

Everyone recognizes the dangers hidden in the third stage. Should we therefore not use all possible ways of preventing this one major complication? Full well is it realized that good conduct of all the stages of labour will lessen the incidence of post-partum haemorrhage. It is all very well leaving matters to nature when the whole structure of our civilization has been a gradual departure from natural living.

During the first stage of labour sedatives are usually given whereas sedatives and/or analgesics are administered during the second stage. Any drug that will shorten the third stage and will cause the uterus to contract and remain contracted, therefore, is of immense value. Ergometrine possesses most of these properties. Unless contraindicated, by the presence of severe toxaemia (ergot may produce spasms) and heart disease, ·5 mg. may be given either intravenously or intramuscularly when the head is crowned, should an expert be doing the delivery; or with the birth of the baby's anterior shoulder, should the accoucheur as yet not be proficient in dealing with the second stage complications (impacted shoulders). Delivery of the foetus should coincide with uterine contraction. The third stage in these cases is, as a rule, not only shortened but relatively bloodless. Let it not be thought that all haemorrhage and long third stages are obviated by ergometrine—far from it. The incidences, however, are greatly lessened. Many obstetricians do not subscribe to the almost routine use of ergot. Their views are respected. Many of these non-subscribers, however, use ergot when post-partum haemorrhage is expected, i.e. when labour has been long or forceps is being applied—*yet* post-partum haemorrhage most often occurs when least expected.

Let us now return to the treatment of post-partum haemorrhage. Should a midwife call you to a patient whose labour she has been attending stating that the patient is bleeding (whether the placenta is in or out of the uterus), the first instruction over the telephone should be: 'Give her ·5 mg. ergometrine intramuscularly. I am coming immediately.' On arrival, always remember to *inspect the placenta* after full clinical assessment and possible management of the patient.

There is still much to learn about the physiology and management of the third stage of labour. The prevention of complications should be our continual aim. The old adage that labour can be said to have been normal once it is well and truly over, is as true today as it has been in the past.

A COMMENTARY ON ANCIENT MEDICINE: IN A LIGHT VEIN

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The title of this article has nothing to do with phlebitis, phlebotomy or intravenous therapy. The humorous, even in history, makes life endurable, and as regards medicine, old or new, is a pleasant interlude to the study of osteology or the incidence of nail-biting in schoolboys.

The study of history is always fascinating, and especially is it so to the doctor when he reads and ponders over ancient medicine. One often forgets that the topmost leaves of the mighty yellowwood tree have for their origin the hidden roots in the earth, where these have been for a thousand years. And so the marvellous and brilliant achievements of modern medicine have been slowly and painfully built up on the labours and thoughts, often in retrospect luc:rous and fantastic, of an ancient colleague of ours some 6,000 years ago. One must admire those 'healers of the sick', most of whom probably laboured conscientiously to the best of their ability and according to the light they had received. But in what a world of total darkness and ignorance about the composition and functions of our body, and the nature of disease, did they grope! Each generation of doctors has at times been inspired by, and has marvelled at, the labours of its predecessors, but has also been amazed and amused at their views and therapy. And likewise future doctors will judge us—till a split atom destroys our world.

Ignorance and superstition have always gone hand in hand. Superstition has been the essence or life-blood of the human race in the dawn of its existence, especially with regard to ill health, disasters and prognostications; it is observed in all primitive peoples and even today is found amongst the most civilized. We must admire the courage of our predecessors of remote ages who, engulfed by these two factors, nevertheless probed in the dark and searched for the truth. One of the most fruitful sources of medical and therapeutic errors, and also in other fields—more so in the past, though it is still common today—was the fallacious reasoning of *post hoc ergo propter hoc*; because event A took place after B, A was necessarily the cause of B. An instance of such reasoning is recorded where a Turk, suffering from typhus fever, in his delirium drank water in which cabbage leaves were being soaked. *Mirabile dictu!* he recovered, and for years afterwards cabbage infusion was used as a sovereign remedy for this disease. A comet appears; a pestilence decimates the population: the comet caused the disaster!

From the pages of the Greek historian Herodotus, an astute observer and delightful writer, who lived some 400 years B.C., one has culled some priceless gems about ancient beliefs and practices. Many things he observed himself; of others he was told.

A King of Egypt had developed eye trouble (probably trachoma), which had blinded him for ten years and which the royal physicians could not cure. The blindness was a punishment for having committed a sacrilege when in his rage he had thrown a spear into the swirling waters of the Nile, which had risen far above its usual level. In his despair he consulted an oracle from a temple. One must admire the skill and astuteness with which many of these oracular words were couched, often obscure and capable of several interpretations. This priest knew that the women of his time observed a very flexible moral code and, as he thought the prognosis pretty hopeless, he advised the King to bathe his eyes with the urine of a chaste woman. He first tried his Queen, and thereafter many women without any success. At last the miracle happened: he was cured. Is it fantastic to think that this particular urine contained an excess of potent adrenocortical steroids? Herodotus

naturally does not mention this remarkable hormone. Today we use this therapeutic agent, but obtained in a less objectionable, easier and definitely more certain manner. Years ago in my time female urine was used as an eye lotion for sties! The happy but sadly disillusioned King collected all the unfaithful ladies (his Queen included) and the unchaste virgins, put them into a room and set the house on fire. Needless to say in true romantic fashion he married the restorer of his sight, and presumably they lived happily ever afterwards. If that King had possessed commercial instincts what a fortune he could have made by patenting and marketing the queenly urine in a land probably harassed by endemic trachoma! One can visualize agents of enterprising pharmaceutical firms peddling this astounding royal remedy throughout the country. We, and especially our womenfolk, living now can truly be thankful that the punishment inflicted by the King on the unfortunate ladies has not been incorporated in our laws, and that this acid test for fidelity and virginity has not been produced in our divorce and law courts.

It will be of interest to all of us, and a contribution to the acrid dispute between general practitioners and specialists, to know that our historian found in Egypt a state of affairs similar to what we have here. (Who said that specialists arose within the last hundred years?) I quote his words: 'The practice of medicine they split up in several parts, each doctor being responsible for the treatment of one disease only. There are in consequence innumerable doctors, some specializing in diseases of the eye, others of the head, teeth, stomach and so on: while others deal with the sort of troubles which cannot be exactly localized.' Obviously the last class was the G.P. of that day. He, as in our time, had to know everything, diagnose and treat obscure diseases, for which he probably got a miserable pittance in comparison with the specialist's fee. His drugs were probably mere placebos, containing harmless or witch ingredients, as described by Shakespeare in the cauldron of the witches of Macbeth. Fortunately he, as well as the specialist, had Nature and Time as invaluable allies. (Are not many of our reputed cures that are attributed by credulous and grateful patients and relatives to our skill and super-knowledge and a host of injections and vitamins etc. due to unknown factors and natural defences of the body—the *vis medicatrix naturae* of the old healers?

In Babylonia there were no doctors: what a scope and scoop for squatters! Invalids were brought out into the streets. Any one passing along *must* stop and ask the sick person what was the matter. He could then offer him advice on his complaint, either from personal experience or observation of a similar trouble in others.

The Egyptians believed that all the diseases (no doubt punishments by the gods excepted) came from the food eaten, and consequently they purged themselves with emetics and clysters every month for three successive days. Herodotus remarks that they were amongst the healthiest people in the world. (Surely no one can boast about Egyptian health today!) He believed that this happy state of affairs was due to the absence of changes in the climate, 'for change and especially change of weather was the prime cause of disease'. Obviously in a mild and constant climate the call on the heat centre and its attendants—vasodilators, constrictors and sweat-glands—as well as the wear and tear of the sympathetic and parasympathetic nerves would be little. But imagine three days of vomiting and purging! One wonders how doctors and business men performed their duties on those days, and what financial losses employers incurred with every one of their workmen absent for three days every month. It would have been a calamitous day if this drastic prophylactic treatment was carried out by all at the same time, unless these days were proclaimed as public holidays; hardly holidays!

In a certain Indian tribe there were no doctors for the simple reason that no one was allowed to get ill. It was a crime against the community, and the death penalty was imposed. Whenever a person fell sick his closest companions killed him because, as they put it, their meat would be spoiled if he was allowed to waste away with disease. 'The invalid naturally protests, but to no purpose; his friends refuse to accept his plea and kill and eat him just the same.' The women eat their women friends. If any one was lucky enough to live to an advanced age (which seldom happened, for most people would have had some disease or other) he or she was offered in sacrifice and devoured. These proofs of true friendship were certainly very economical: no doctors' bills, no hospital fees, no cemeteries with expensive coffins; moreover the butcher's account was considerably reduced.

An Egyptian King, a captive at the court of Cambyses, son of the great Persian King Cyrus, was caught stirring up trouble amongst his own people; 'he was made to drink bull's blood and died on the spot. And that was the end of Psammenitus!' There must have been a very severe incompatibility between the blood of this gentleman and that of the bull. One wonders if the phenotype of his blood was perhaps group $O_1 R_1 R_2$ (C De/c D E), M Nss, P-, Fy^a+, Lu-, K K. This formula is enough to kill any ordinary Egyptian, even a King.

Herodotus heard interesting tales about the Ethiopians on the coast of the Indian Ocean, related by spies whom the Persian King Cambyses had sent to that country. They were the tallest and best-looking people in the world. They all lived to the age of 120 or more; their food was only boiled meat and milk. There was a spring, the water of which smelt like violets and made a man's skin glisten as if he had washed it with oil. The water lacked density and nothing would float on it. The constant use of this water must be the cause of the longevity. Further the semen of these men was black like their skins—a peculiarity also reported from the Indian tribes mentioned above. Well, well! Was this due to an inborn error of metabolism (in Garrod's phrase) which caused a disturbance of the pigment-forming hormone; or was it a waste black pigment excreted by an unusual route? I suppose there was something logical about this belief: a black man must of necessity have a black discharge to produce a black-skinned offspring. A similar reasoning would make a black hen lay a black egg; and the yellow races would have yellow semen. In intermarriages one could expect, from the Mendelian laws of heredity all sorts of testicular or prostatic discharges according to appearance—a veritable variety of semen in technicolour! At this stage it is perhaps appropriate to record our historian's dryly humorous and sensible remark: 'My business is to record what people say, but I am by no means bound to believe it.'

There are many other fascinating tales about the customs and beliefs of peoples who lived in Northern Africa and Asia which have a bearing on medicine and sickness; also of strange animals and their habits in producing offspring. We also, fortunately, are by no means bound to believe these stories.

HOSPITAL FOR ALCOHOLICS

Hospital for Alcoholics. The Cape Provincial Administration has decided to establish a hospital for alcoholics at Rondebosch, Cape. The hospital will be the first in South Africa to be run by a provincial administration and it will probably be opened in February 1959.

The hospital will be included in the medical services rendered jointly by the Provincial Administration and the staff of the University of Cape Town. Patients will be assessed on their income and size of their families and pay in the ordinary way. As the hospital will be in a European area, only Europeans will be admitted, but if it is a success the scheme will no doubt be extended for Coloured people. The new hospital will be an experimental hospital. The attitude of the public will be crucial and will have much to do with the success of the venture. There will be 24 beds for men and 6 beds for women. During the day most of the patients will spend their time walking in the grounds and taking part in group discussions.

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EDITORIAL : VAN DIE REDAKSIE

THE HAGEMAN FACTOR

People who perform silly tests must expect that sometimes they will get silly answers. In certain hospitals a patient who is admitted for almost any disease may be put through the diagnostic sieve. Since there is no shortage of laboratory tests this work up may only be limited by the patient's ability to pay for these tests. In many cases an extensive laboratory investigation is essential for diagnosis and treatment; occasionally a rather 'unnecessary' investigation yields surprising results. One such example led to the discovery of the Hageman factor.

There are apparently patients who have a very prolonged coagulation time although they give no history of severe haemorrhage. The relative insensitivity of the clotting time has been known for a considerable time. Many patients who are undoubtedly bleeders have a normal coagulation time. Probably if one were to investigate a large number of people who state that they bleed excessively, only a minority would be found to have a prolonged coagulation time. Even in the group of unquestioned haemophilics there are a considerable number who have normal coagulation times.[1,2] But one does not normally expect to find a prolonged coagulation time in a person who has no bleeding tendency. Yet cases such as this have been described.[3-5] In all these cases 'routine pre-operative' coagulation times were performed and the results were received with surprise. In none of these cases had excessive bleeding been a feature of the clinical story. Some of the cases had had major surgical procedures performed without incident before the test was made, and after the test surgery was also safely performed. The fact that the clotting time can be lengthened to 3 times the normal without indicating a significant haemorrhagic tendency raises the suggestion that the test as usually performed may have no practical significance, and that glass containers may introduce serious irrelevance. This would certainly be true if the discrepancy was encountered with any degree of frequency. It is, however, excessively rare, and pathologists who have time to look for such cases probably need to be provided with more useful occupation![6] However, investigations carried out on the blood of patients presenting this rare phenomenon have provided evidence of very great interest both from a practical and theoretical point of view.

The first patient in whom this phenomenon was investigated in detail was a certain Mr. Hageman; hence the name. The condition is apparently familial and a similar deficiency has been discovered in the blood of Peking ducks.[7] The blood of these patients is apparently insensitive to the effect of glass contact. That glass contact affects the coagulation of the blood has been known for a considerable time. In 1901 Bordet and Gengou[8] found that the clot-promoting effect of glass could be shown even after the bulk of the blood cells had been removed, and this work has been amply confirmed.[9-12] This clot-promoting effect of glass was demonstrable also in plasma which had been absorbed with barium sulphate (or aluminium hydroxide) and which had been heated to 56°C for 30 minutes.[11,12] Plasma so treated was devoid of most clotting factors but still contained the Hageman factor and the so-called Rosenthal's factor (PTA). Why glass should act in this way is not clear, but Ratnoff and Rosenblum[7] believe that an inhibitor is removed by contact with glass. They believe that glass exerts its clot-promoting effect by releasing Hageman factor from inhibition. Plasma from patients (or animals) deficient in Hageman factor impedes the clot-promoting effect of glass on normal plasma. However, once glass had been allowed to exert its effect on normal plasma the addition of Hageman-deficient plasma does not appreciably inhibit clotting. Margolis[12] reported that the accelerator which appeared when plasma was treated with glass was labile, since its activity appeared to be destroyed by some agent in the plasma. The concept is that the fluidity of the blood in the blood vessel is maintained by the presence of inhibitory substances directed against the Hageman factor and that exposure of blood to glass removes this inhibitor. The uninhibited Hageman factor then initiates or promotes clotting.

This clot-promoting effect of glass is present in plasmas devoid of antihaemophilic factor, Christmas factor, plasma thromboplastin antecedent (PTA), factor V, factor VII and Stuart factor, as well as in patients treated with dicoumarol or patients with liver disease. It is not implied that glass cannot act on factors other than Hageman factor (especially the blood platelets), and even blood from patients with Hageman-factor deficiency may show accelerated coagulation on agitation with glass.[7]

These concepts are important in at least two ways. The first is technical. In an investigation of the calcium clotting time in a patient with possible bleeding disease it is essential to standardize at least the presence or absence of blood platelets and also the amount of glass contact. Margolis[13] has recently introduced a kaolin clotting time which does just this, and he reports that the clotting time of recalcified plasma then gives reproducible results and is of value in the diagnosis of patients with bleeding disorders. But an even more important concept might arise from these phenomena. Any factor which plays a part in maintaining the fluidity of blood in blood vessels is obviously of immense potential importance in the study of disease processes. The concept of the pre-thrombotic state is well established. Numerous studies have been made in an attempt to provide laboratory confirmation of a tendency to form thrombi in blood vessels. In potentially thrombotic states such as, for instance, occur after operations there is an increase of plasma fibrinogen and of blood platelets. But we remain unable to recognize the patient who will develop the vascular thrombus. To be able to do so would be of immense prac-

tical importance and it is conceivable that studies such as those mentioned above may throw some light in this rather obscure field.

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DIE MIGRAINEGESTELDHEID

Een van die chroniese toestande wat taamlik algemeen voorkom en wat dikwels omrede van sy wisseling van vorm misgekyk word, is migraine. Dit is 'n toestand wat van kleins af kan voorkom en dikwels lewenslank kan duur, maar wat baie keer ook in sy natuurlike geskiedenis en verloop belangrike wisselvorme en vormveranderinge toon.

By kinders wat onderhewig is aan migraine, is daar soms 'n neiging om ‚naar-hoofpyne' te kry met die grootste nadruk op die naarheid. Dit is om hierdie rede dat migraine in hierdie vorm gewoonlik gewyt word aan eetvoorvalle en versteurings van spysvertering. Omdat hierdie soort ‚spysverteringsaanvalle' sporadies voorkom en van verbygaande aard is, word daar gewoonlik nie veel meer aandag daaraan geskenk nie as om te sê dat die kind versigtig moet wees met wat hy eet.

Hierdie aanvalle van ‚naar-hoofpyn' by kinders wat onderhewig is aan migraine gaan dikwels in die middelste jare—tussen 20-45—oor in die tipiese paroksismale migraine-aanval. So 'n aanval word dan gewoonlik voorafgegaan deur gewaarwordinge wat die vorm kan aanneem van ligflikkeringe, versteuring van gesig (gewoonlik van die een of ander deel van die gesigsveld), parestesie, fotofobie en oorgevoeligheid vir geluide en gerase, gevoelens van verwyderd te wees, ens. Vyftien tot 20 minute later volg dan gewoonlik die bekende skeelhoofpyn wat op hierdie stadium gewoonlik die opvallendste kenmerk van die aanval is, ofskoon naarheid en braking dikwels met die hoofpyn saamgaan. Die aanvalle kan lig wees of in hewige vorm voorkom sodat die pasiënt volstrek onbevoeg is om enigiets te doen.

Teen ongeveer die ouderdom van 40-50 tree dikwels die volgende faseverandering in waarby die naarheid en die hoofpyn van die eerste twee fases op die agtergrond tree en die sogenaamde migraine-ekwivalente prominent op die voorgrond kom. As die dokter nie baie goed bekend is met die geskiedenis van die pasiënt nie, is dit gewoonlik hier waar die basiese migrainegesteldheid misgekyk kan word.

Ekwivalente aanvalle kan nou voorkom sonder hoofpyn en daar is gewoonlik die volgende bygaande simptome: Ongewone prikkelbaarheid, 'n gevoel van verwyderdheid, 'n gevoel van gejaagdheid, besondere honger en oordrewe eetlus, gaperigheid en dwarrelende gedagtestrominge met helder beelde en herbelewenisse uit die verlede. Hierdie ekwivalente verskynsels kan ook in ligte graad voorkom sodat die pasiënt self die toestand nie eers herken nie (en ander mense dink hy is net moeilik of prikkelbaar of temperamenteel), of die ekwivalente verskynsels kan in so 'n erge graad voorkom dat die hele ondervinding skrikwekkend vir die pasiënt is en hom vreesbevange laat met twyfel oor sy eie geestesgesondheid.

Dat migraine hierdie soort verloop het in die drie fases wat ons geskets het, is taamlik algemeen, maar dit kom nie noodwendig so voor nie. Migraine is 'n toestand wat by uitstek deur vormwisseling gekenmerk word. Dat die toestand ook in 'n sekere soort persoon voorkom, ly geen twyfel nie. Die migrainelyer is dikwels 'n fynbesnaarde, intelligente persoon wat die geneigdheid toon om met die aanvalle van hoofpyn ook spastiese toestande van die kolon te kry.

Tot so ver het ons nou nog net die konstitusionele, idiopatiese migrainegesteldheid beskryf. Die moontlikhede van ander veroorsaking van migraineagtige hoofpyne soos byvoorbeeld, subarachnoïde bloedings, ens., moet nie uit die oog verloor word nie.

Wat die behandeling van die toestand betref, moet ons dink aan voorsorgsmaatreëls en aktiewe terapie. Onder die voorsorgsmaatreëls moet die volgende genoem word: Waak teen emosionele spanning, besondere koue, lank staan, die glans van skerp lig, die eet van kos wat 'n aanval van spastiese kolon kan veroorsaak, en moontlik ook die eet van sjokolade en ryk varkvleis. Omdat glans so 'n algemene prikkel is, is dit goed vir migrainelyers om 'n ligte getinte glas te dra in 'n sonbril, of 'n ligte getinte lens as hulle brildraers is.

Wat die spesifieke terapie by aanvalle betref, hang alles af van die aard van die aanval. Daar is veral drie middels wat afsonderlik of in kombinasie gebruik kan word, wat per inspuiting of deur die mond gegee kan word, en wat baie effektief is, veral as hulle vroeg in 'n aanval toegedien word. Die basiese bestanddele van hierdie middels is ergotamien, kaffeïen en fenobarbitone. Hierdie middels is vandag in baie preparate verkrygbaar. Veral middels wat ergotamien bevat, behoort, om die maksimale uitwerking te hê, toegedien te word sodra daar aanmannings van die aanval is en nog voordat die hoofpyn te voorskyn kom—indien dit moontlik is.

Omdat migraine chronies van aard is, moet daar altyd gewaak word teen die gebruik van gewoontevormende middels vir migrainelyers. Die regte benadering van die toestand, die sorgvuldige tref van voorsorgsmaatreëls en spoedige aktiewe terapie kan baie doen om die lewe van die migrainelyer minder onaangenaam te maak.

'GENERAL PRACTICE SERIES'

The theory and practice of modern medicine has advanced with such rapid strides that it would be no exaggeration to say that the whole face of medicine has changed. This change has been especially noticeable in the fields of anaesthetics, operative procedures and in the drug treatment of disease, but it has also had a very important bearing on the

practice of medicine by the general practitioner. In spite of this, however, it remains true to say that we have not completely outgrown the usefulness of the time-honoured art of clinical medicine.

In order to try to give some guidance in the numerous problems of management and treatment with which the modern general practitioner is confronted, and in order to arrive at a re-evaluation and a synthesis between proved clinical methods of treatment and the newer scientific approaches, we have invited distinguised contributors to provide a series of signed articles which it is hoped will be helpful to the practitioner. These articles will be published at fortnightly intervals.

'ALGEMENE PRAKTISYNSREEKS'

Die teorie en die praktyk van die moderne medisyne het met sulke rasse skrede vooruitgegaan dat dit geen oordrywing sal wees nie om te sê dat die hele medisyne wesenlik verander het. Hierdie verandering is veral merkbaar op die gebiede van die narkose, operasietegnieke en die behandeling van siektes met medisyne, maar dit het ook 'n baie belangrike neerslag gehad op die mediese praktyk soos beoefen deur die algemene praktisyn. Ten spyte hiervan bly dit egter tog nog waar om te sê dat ons nog nie die waarde van die ou beproefde kuns van die kliniese medisyne ontgroei het nie.

Met die doel om te probeer om leiding te gee ten opsigte van die baie probleme aangaande die hantering van pasiënte en behandeling waarmee die algemene praktisyn te doen kry, en in die hoop om tot 'n herwaardering en 'n sintese te geraak tussen kliniese metodes van behandeling en die nuwere wetenskaplike benaderings, het ons 'n aantal bekende medewerkers gevra om 'n reeks getekende artikels te skryf wat, na ons hoop, van nut sal wees vir die algemene praktisyn. Hierdie artikels sal al om die ander week gepubliseer word.

SOME CLINICAL ASPECTS OF DIABETES WITH RELATION TO INSULIN AND TOLBUTAMIDE

A DISCURSIVE SURVEY WHICH ASKS SOME QUESTIONS BUT ANSWERS NONE*

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A. VARIETIES OF DIABETES

I should like to start by reaffirming the clinical separation of diabetes mellitus into 3 types, severe, mild, and secondary. 'Severe' refers to the usually young, growth-onset, ketosis-prone individual, who needs insulin to live. 'Mild' refers to the usually older, maturity-onset, often obese person, who does not become ketotic when insulin is omitted, although he (or more usually she) may be relatively insensitive to insulin and require 100 or even more units to produce any obvious effect on her carbohydrate metabolism. The dose of insulin being used is no criterion of the severity of the diabetes. Severe and mild diabetes are sometimes called 'pancreatic' and 'extrapancreatic' respectively, on the assumption that the disease in the first case is due to destruction of pancreatic beta-cells, and in the second to anti-insulin factors which may or may not be hormonal. However, insulin antagonists have been found in the blood-stream of the severe diabetic rather than in the mild case, while islet-cell damage probably always occurs in the pancreas of the mild diabetic, in whom both Ogilvie[1] and Gepts[2] have found an invariable reduction in amount of islet tissue. Vallance-Owen[3] has shown that the insulin-like activity in the blood-stream of the mild diabetic is of the same order as in a fasting normal person. This, however, does not mean that the pancreatic activity is normal; far from it, since an induced rise of blood sugar in the normal to the height of that in the mild diabetic produces a several-fold increase in plasma-insulin activity. Thus the mild diabetic possesses a pancreas which is unable to respond normally to the stimulus of a raised glucose level.

It may be noted that the terms 'mild' and 'severe' refer only to the evidence of abnormality of metabolism; both types may develop the same crippling or killing vascular complications, both may present similar inheritance patterns and occur together in the same family, and both may give evidence of their latent existence by the same sort of abnormal obstetric history and embryopathy in the prediabetic phase. It is also true that a mild diabetic may develop ketosis under the influence of certain forms of stress, while occasionally a severe diabetic reverts to the mild form. I have twice seen patients in hospital in diabetic coma, without any known precipitating factors, who were normoglycaemic some months later without insulin. Nevertheless the clinical differentiation of the main primary types of diabetes is extremely important and remarkably often neglected. For instance, figures concerning the clinical use of sulphonylureas with regard to age of patient, insulin dosage and so on are quite useless unless the two diabetic groups have first been clearly separated.

'Secondary' diabetes refers most obviously to that which follows chronic destructive pancreatitis, haemochromatosis or total pancreatectomy. The diabetes which occurs during pregnancy, staphylococcal infections or acromegaly, or with moderate doses of glucocorticoids, is not truly secondary, but rather the unmasking of an underlying latent or pre-diabetic state. Fig. 1 shows a corticoid diabetic (European male aged 35, suffering from gout) becoming apparently normal after cessation of treatment, but again diabetic—this time permanently—a few months later.

* Being a contribution to a symposium on 'The mechanism of action of insulin and other hypoglycaemic substances', London, 10 September 1958, under the auspices of the British Insulin Manufacturers.

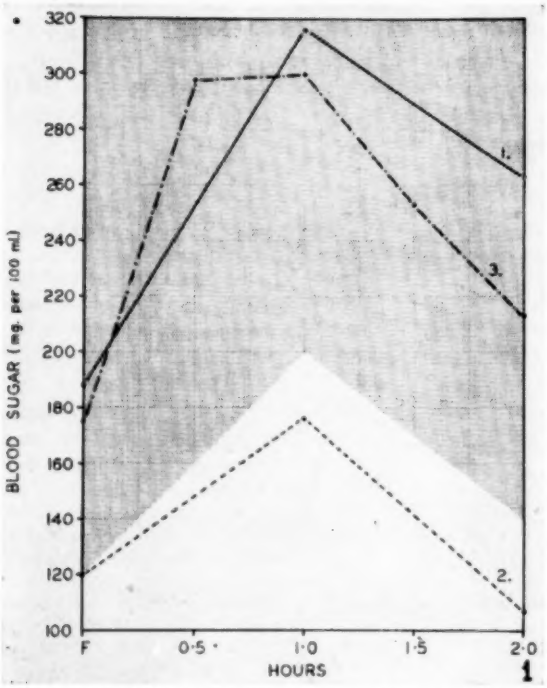

Fig. 1. 'True' diabetes uncovered by cortisone. 1. Diabetic after 4 months on cortisone. 2. Normal curve off cortisone. 3. Eight months later, no more cortisone: again diabetic. (Glucose-tolerance curves using 50 g. of glucose by mouth.)

PREDIABETES AND VASCULAR DISEASE

Many of us believe that, strictly speaking, diabetes begins at birth, that the basic abnormality, whatever it may be, is present for the whole of the lifetime of the diabetic, although the overt carbohydrate defect may not be manifest until middle age. Time precludes discussion of the arguments in favour of this belief, beyond my remarking that the elderly diabetic who attends your clinic may give a history of large babies and stillbirths which extends back 30, 40 or 50 years into her prediabetic past. Furthermore we consider that the specific vascular abnormalities of diabetes are not 'complications' but are an integral part of the syndrome, inherited together with the metabolic disturbances. Usually they appear later than the hyperglycaemia, but sometimes before it. They are not a consequence of hyperglycaemia, and normalization of the latter, even in the very mildest cases, is no safeguard against their occurrence. It would, however, appear that they are closely connected with pancreatic damage, since true secondary pancreatic diabetes has occasionally led to the later development of specific retinopathy or nephropathy.

These various observations are extremely important, since they appear to intimate that a defect of pancreatic islet tissue may produce a diabetic vasculopathy which is not dependent upon hyperglycaemia. Indeed we have perhaps been overobsessed with hyperglycaemia as the primary feature of diabetes. Insulin has an action in fat metabolism which may well be more important than its more obvious effect on carbohydrate. It is closely related to the level of the non-esterified fatty acids, and stimulates rapid metabolism in fatty tissue. It has now been shown to exert an influence on amino-acid metabolism.[4] Might it perhaps have an essential protective effect on blood-vessel walls? A future diabetic might be born with a relatively inefficient beta-cell mechanism, not so defective that our rough yardsticks of definition based on glucose tolerance can detect it, but bad enough that throughout the years certain blood vessels in certain places become progressively damaged. The special stress of pregnancy may indicate the latent deficiency by producing temporary carbohydrate intolerance in the mother, death or excessive size of the foetus, and hyperplasia of the foetal islets of Langerhans.

INSULIN AND PREDIABETES

This hyperplasia of the islets of Langerhans in the foetus of a diabetic or a prediabetic mother is a remarkable phenomenon. Table I shows the relative proportions of islet tissue to the whole pancreas which Woolf and I[5] found on examination of 108 pancreases from stillborn infants. Fig. 2 illustrates a pancreas from the foetus of a prediabetic mother (B) compared to a normal at the same magnification (A). From the small amount of suitable material available to us, it seems that not only are the islets large and increased in number, but they contain an unusually high proportion of beta cells, and these beta cells contain an unusually great concentration of granules. If granules really represent insulin, then it would appear that the pancreas of such a foetus may contain up to 30 times as much insulin as normal. What could be the cause of this beta-cell hyperplasia? It is not hyperglycaemia, because it occurs in the absence of hyperglycaemia in the prediabetic. Growth hormone and glucocorticoids have been much discussed in connection with the diabetic embryopathy, but there are cogent arguments against either of these as the sole or primary villain of the piece. Might not the maternal lack of pancreatic reserve during pregnancy militate through some mechanism other than hyperglycaemia to allow a compensatory hypertrophy of the infant's islets? Now the excessive insulin produced by the infant might itself act as a 'growth hormone' in its own intra-uterine development. Could this possibly account for the excessive size of the diabetic's baby?

TABLE I. PROPORTIONS OF ISLET TISSUE IN PANCREASES IN DIFFERENT GROUPS (EXPRESSED AS MEAN PERCENTAGES OF TOTAL PANCREAS)

Control stillbirths	1·3%
Stillbirths of diabetic mothers	6·5%
Stillbirths of prediabetic mothers ..	7·5%
Stillborn with erythroblastosis foetalis ..	7·1%

If this story has any truth, then insulin might be expected to prevent the diabetic vascular abnormalities, the foetal and obstetric abnormalities, and possibly the actual development of overt diabetes in a prediabetic. Certainly, and unfortunately, insulin will not prevent all vascular disease in diabetics, but there is much evidence that when it is given in such a way that good control of glucose metabolism is achieved then the liability to vascular lesions is, in general, considerably lessened. Figures from our own clinic indicate that the incidence of retinopathy and neuropathy in poorly controlled diabetics was double that in diabetics whose control was good or excellent. Now if our belief regarding

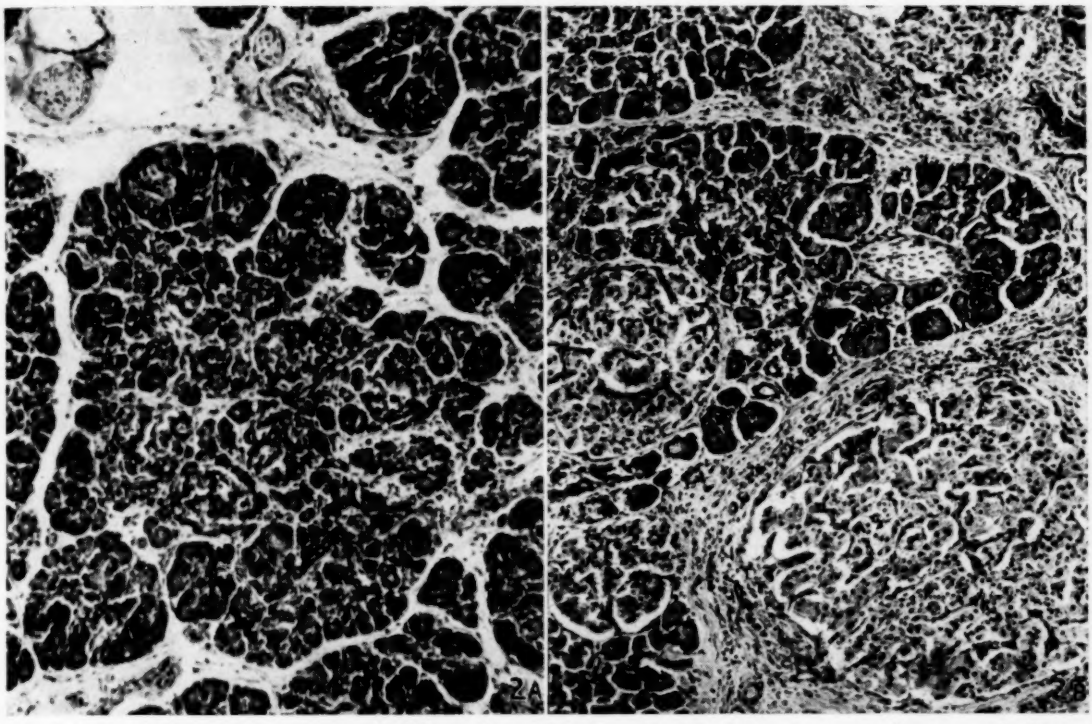

Fig. 2*A*. Photomicrograph of pancreas of stillborn of normal mother. *Fig.* 2*B*. Photomicrograph of pancreas of stillborn of prediabetic mother.

the presence of diabetes from birth is true, it is surely plain that insulin given only when overt carbohydrate defects are present would not be expected to prevent a lesion which has already been developing for a long time. If we could give insulin early in the prediabetic phase, might we not then prevent the vascular disease? This, of course, is not known, but it is worth considering. Methods used in diagnosing prediabetes I will not consider here, but it may be mentioned that certain family histories make a diagnosis of pre- or latent diabetes mandatory; for instance when both parents are diabetic, when an identical twin is diabetic, or in such a family as is shown in Fig. 3. Prof. Hoet[6] has indeed some evidence that insulin given to a prediabetic woman during pregnancy will prevent the development of severe congenital abnormalities, while Wilkerson[7] from Boston has shown that the same treatment will tend to reduce the birth weight of the foetus. Further, Hoet[5] has pointed out that the child of a known diabetic mother being treated with insulin seldom becomes diabetic under the age of 10, whereas almost all mothers of children who become diabetic under the age of 6 are untreated latent or pre-diabetics. Insulin given during pregnancy may thus have a protective action on the child. Is it just possible that insulin given to children in diabetic families would prevent diabetes entirely? It protects the pancreas of animals against alloxan.

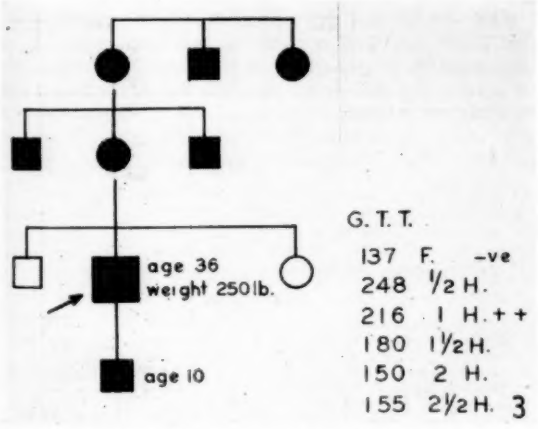

Fig. 3. The blacked-in figures represent diabetics. The proband (arrowed) had a diabetic son, mother, and grandmother. There was no diabetes on his wife's side. He did not know he was diabetic, but the family history led to his having a glucose tolerance test; result as given.

PROPHYLAXIS WITH INSULIN OR ORAL SULPHONYLUREAS

It is admittedly impracticable to suggest that babies or even adults should be stuck with a needle every day without a great deal more evidence of its value. Could the oral sulphonylureas take the place of insulin in prophylaxis? It is very important that we should know the mode of action of these substances before embarking on such a programme. If their

major action is a stimulation of beta cells with the production of insulin, then there are two ways in which their use in prophylaxis would differ from that of insulin. First it might be presumed to overtax the already inefficient beta cells, rather than to rest them. The whole idea of continued stimulation producing permanent structural damage to the beta cells is, however, rather nebulous, has been too readily accepted, and finds no proven counterpart in the rest of the endocrine system, nor indeed in other organs of the body. Gepts,[2] furthermore, has found that the damage to islet cells in elderly patients treated with carbutamide or tolbutamide is no greater than that seen in a group of similar untreated or insulin-treated diabetics, while in some he found actual regeneration of apparently active beta cells. The second difference between tolbutamide and *exogenous* insulin is that the *endogenous* insulin which is formed under the influence of tolbutamide would be liberated into the portal system, and so might really act more physiologically than insulin injected peripherally. Thus Madison and Unger[8] have shown that insulin administered intraportally in dogs produces a much smaller arterio-venous glucose difference than when systematically introduced, suggesting a lesser effect on muscle and probably a greater hepatic effect. Tolbutamide does not appear to be toxic in other ways—in fact it seems to be about the most non-toxic drug ever produced. Theoretically it might appear inadvisable to give it to a prediabetic or diabetic during pregnancy, since we have evidence that the beta cells of the foetus are already being excessively stimulated. Although tolbutamide might be called simply a drug of convenience in the treatment of the established mild diabetic, it may yet be of true value if used early enough, especially in the prediabetic.

The advent of the antidiabetic sulphonamides has taught us more about diabetes as seen in the clinic. In the trial of carbutamide and tolbutamide which we carried out, we made it a rule to withhold all insulin from our mild diabetics for several weeks before starting the new drug, in order to obtain a proper control base-line. We were surprised to find the number of patients whose carbohydrate metabolism was as well controlled on diet only as it had been with insulin also. In fact in a few the control was actually better without insulin. This has led to a considerable reduction in the number of patients taking insulin in our clinic. Furthermore it clearly indicates that any trial of an oral preparation in diabetes is valueless without periods during which no drug at all is taken. Although insulin was apparently being given unnecessarily to the above-mentioned patients, yet perusal of their previous records frequently indicated that it had been prescribed to good purpose when originally administered, 2, 3, or 5 years before. This might be interpreted in 2 ways; either the disease in its natural course had become milder or, as I prefer to think, the insulin had really had a partially curative effect.

Some clinics in Britain have been strongly advocating a return to the use of soluble insulin alone for the control of all severe, insulin-dependent diabetics. They claim not only better carbohydrate control, but also a greater protection against vascular disease than is afforded by long-acting insulins. I do not consider they have proved either point, and it must be remembered that the long-acting insulins were introduced largely because 2 injections of soluble a day were inefficient in diabetic control. We have found, after some 5 years experience, that the lente group of insulins are generally satisfactory and we consider that their wholesale abandonment would be a retrograde step unless further evidence is forthcoming to warrant such a procedure.

PRIME IMPORTANCE OF VASCULAR DISEASE

Despite the above arguments I do not wish to overemphasize the potential protective value of insulin in diabetes. Now that coma is a relatively minor problem, the importance of diabetes does not lie in its carbohydrate control but in its vasculopathies—retinopathy, neuropathy (assuming that this is basically vascular), nephropathy, and coronary and peripheral vascular disease. The blood sugar of itself is a matter of little importance. Although good control of diabetes as measured by glucose estimations may partially prevent the specific diabetic vasculopathies, yet it has little or no effect on the development of coronary heart disease.[9] We have observed this in figures from our own clinic. Normally, pre-menopausal women are considerably protected from the clinical manifestations of coronary atheroma by the very fact of their womanhood. In this respect diabetes abolishes the advantage of being born a woman. If, however, the coronary arteries can really be protected by a low intake of saturated fat, then surely the very first place in which this type of diet should be advised is the diabetic clinic? If the carbohydrate intake is increased and socalled 'control' rendered more difficult, is there any evidence at all that this would matter? It seems to me that many 'diabetic diets' being used today are probably atherogenic. As recently as June 1958 a report of the Council on Food and Nutrition (US) recommended that 40% of calories in a diabetic's diet should come from fat. Admittedly a small face-saving, and rather ingenuous, rider was added: 'with unhydrogenated vegetable oils substituted to a considerable extent for hard cooking fats'. Comment on the utter inadequacy of such advice should be unnecessary. There is certainly less evidence that the *specific* diabetic vascular lesions might be reduced by a lowered fat intake, but this is something that must be investigated.

CONCLUSION

I think that all this may be summarized by the suggestion that insulin, and even possibly the oral sulphonylureas, may have a greater potentially protective action in diabetes than we realize, but that drugs alone are not likely to solve the real clinical problem of diabetes—the vascular one. We still do not know how best to treat diabetes and are only just beginning to think about preventing it.

I should like to take this opportunity of thanking the British Insulin Manufacturers and especially Dr. F. Wolff for arranging the symposium; also Prof. J. F. Brock and Dr. J. H. Sheldon for reading this manuscript. Drs. N. Woolf and J. A. H. Campbell are responsible for the photomicrographs of the islets (Fig. 2).

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DIABETIC RETINOPATHY DEVELOPING IN TWO CASES ON TOLBUTAMIDE THERAPY

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A great deal of interest has been stimulated in the treatment of diabetes mellitus as the result of the introduction of the sulphonyl ureas during the past 3 years. One of these substances (tolbutamide, D 860), has proved effective[1, 2] in over 40% of the middle-old-age group. The patients who have responded to treatment have been kept under good control so far as blood sugar and urine estimations have shown. They have remained symptom-free and have felt well. However, the great challenge in treatment of diabetes still remains that of the occurrence of the late manifestations of the disease. Vascular changes, hypertension, albuminuria, retinopathy, possibly cataract, and Dupuytren's contraction have all been described in this category.

Even where good control of diabetes has been maintained with insulin therapy, the late manifestations have ultimately appeared. It was hoped that with tolbutamide therapy this would not occur. While realizing that a long period of time must elapse before it will be possible to evaluate the true effects of tolbutamide therapy, we feel that it is important to report any late manifestations occurring during the course of treatment.

One of us (L.M.) has made repeated ophthalmological examinations of tolbutamide-treated patients at the Diabetic Clinic, particular attention being paid to the development of retinopathies or other ocular changes occurring during the course of therapy. As a result it is now possible to record 2 cases in which retinopathies have occurred during treatment.

CASE 1

Mrs. I.J.C.M., aged 61 years, presented with diplopia to the right due to a right external rectus palsy. On investigation she was found to be diabetic, with a heavy glycosuria and a fasting blood sugar of 257 mg. per 100 ml. On diet plus 15 U. of lente insulin the patient became asymptomatic and the rectus palsy disappeared. Her fasting blood sugar, however, still remained high, being 245 mg. per 100 ml. both in July and September 1956. The fundi remained normal throughout.

In October 1956 she was transferred to tolbutamide therapy. Her blood sugar responded immediately and has remained at near normal limits since that time (Table I). In spite of the marked improvement in the diabetic state and the fact that she was perfectly well, gradual changes occurred in the eye grounds from January 1958 onwards. At first only a few micro-aneurysms and occasional punctate and blotch haemorrhages made their appearance but within a matter of a few months the condition had worsened considerably until it was that of an advanced retinopathy with micro-aneurysms, punctate and blotch haemorrhages and discrete exudates throughout both fundi. In June 1958 a slight improvement in the retinopathy was noted, but the condition still remain very marked.

TABLE I

Date	*Blood Sugar*	*Fundi*
*28/ 6/56	257	Normal.
*27/ 7/56	245	
*27/ 9/56	245	
27/11/56	126	
11/12/56	121	
9/ 1/57	131	
7/ 2/57	115	
26/ 2/57	91	
29/ 3/57	81	
3/ 5/57	112	
24/ 6/57	134	
5/ 7/57	122	
2/ 8/57	87	
30/ 8/57	103	
27/ 9/57	110	
8/10/57	135	
19/11/57	95	
10/12/57	104	
10/ 1/58	95	Many micro-aneurysms and punctate haemorrhages, occasional blotch haemorrhages.
31/ 1/58	103	
7/ 3/58	105	Marked deterioration.
1/ 4/58	116	
2/ 5/58	101	
14/ 5/58	100	Micro-aneurysms, punctate and blotch haemorrhages, discrete exudates.
21/ 5/58	77*	
28/ 5/58	83*	
30/ 5/58	102	
27/ 6/58	119	

Blood sugar expressed in mg. per 100 ml. blood. All samples taken fasting except those marked *, which were random samples taken 3 hours after a meal.

CASE 2

Mrs. F.E., aged 68 years. Diabetes mellitus was discovered in June 1957, her first symptoms being mistiness of vision, thirst and loss of weight. Early lenticular changes were present in both eyes; although arteriosclerotic changes were seen there was no evidence of diabetic retinopathy. A further independent ophthalmic opinion 3 months later confirmed the first findings.

Tolbutamide therapy was commenced on 13 June 1957. It will be seen in Table II that although the blood sugar was high originally there has been a gradual drop to near normal limits. Control has not been so good as in Case 1, but the blood sugars have been returning to near normal levels and this patient is under effective diabetic control. In spite of her improved condition and the disappearance of all symptoms, retinal changes have appeared.

TABLE II

Date	*Blood Sugar*	*Fundi*
13/ 6/57	368	Early lenticular changes. Fundi normal except for arteriosclerosis.
2/ 7/57	204	
9/ 7/57	161	
6/ 8/57	308	
15/ 8/57	195	
5/ 9/57	167	
3/10/57	151	
29/10/57	168	
26/11/57	120	
24/12/57	125	
17/ 1/58	139	Many micro-aneurysms and punctate haemorrhages plus occasional blotch haemorrhage.
18/ 2/58	129	
17/ 5/58	140	
20/ 6/58	112	

All samples taken fasting.

The first signs of the changes were noted in January 1958, when micro-aneurysms and punctate and blotch haemorrhages were discovered. These have remained constantly present during the past few months.

DISCUSSION

Two diabetic cases are described who have developed retinopathy while under effective control on tolbutamide therapy. In the one case diabetes has been present for a period of 2 years and in the other for a period of 7 months. In case 1 control of the diabetes has been very good on a regime of tolbutamide plus diet. In spite of this, retinopathy has appeared and advanced rapidly. In case 2 the control has not been as good as in the former, but there has been no glycosuria and the blood sugar has not been markedly raised.

Retinopathy is one of the very distressing late mani-

festations of the disease. While no claims have been made that tolbutamide would prevent the onset of this condition or other late manifestations of diabetes, it was hoped that effective control with this remedy might have this result. The 2 cases described, however, suggest that this is not the case.

The high incidence of retinopathy in diabetes is well known. The longer the duration of diabetes the higher would appear to be the incidence. Waite and Beetham[4] state that 59% of their patients with diabetes of 15 years' standing or more have retinopathy, and Wagener[5] that 60% of more than 10 years standing show a similar condition. Whittington and Lawrence[6] state that 75% with diabetes of 20 years' standing or more show retinal changes, and Ballantyne[7] found 31·7% of cases with retinopathy among 561 diabetics of all ages. Wagener[8] gives a figure of 30·6 for diabetic retinopathy among patients of all ages. One of us (T.S.)[3] found 58 cases of retinopathy among 231 diabetics, 14 of whom showed the condition 0-5 years after the diabetes had commenced.

It is evident that retinopathy may occur early in the course of diabetes, and Wagener[8] has pointed out that in occasional cases of retinopathy the only evidence of diabetes is a positive glucose-tolerance curve. He has suggested that 'a primary disturbance of carbohydrate metabolism is requisite to the institution of the retinopathy, but that this disturbance need not be sufficiently severe to cause permanent elevation of the blood-sugar levels, and that some secondary defect of metabolism may be the actual cause of the retinopathy'.

On the other hand, Fisher[9] states that the principal factor responsible for the development of diabetic retinopathy is long-standing hyperglycaemia, and the best method available at present for the prevention of the degenerative complications of diabetes is exact control of the diabetes through training of the diabetics themselves. Dunlop[10] maintained 39 diabetics on a 'free diet' for 9 years and degenerative complications, including retinopathy, developed in 14 (36%). In a group of 15-31 years duration he found retinopathy in 44% of those with good control and in 79% of those with poor control. He has decided that careful control and aggressive treatment of the diabetes over the years are most important factors in the prevention or postponement of diabetic degenerative lesions.

It is therefore controversial whether good control of diabetes is the answer to the problem of the prevention of retinopathy and the other diabetic degenerative lesions.

Close similarity has been found between the lesions in diabetic retinopathy and those in the Kimmelstiel-Wilson syndrome. Further, both the retinopathy and the renal lesions have been produced in alloxan-diabetic animals by injecting corticotrophin.[11] It has been observed in our clinic that patients experiencing emotional upsets (e.g. the loss of a husband) have had a marked exacerbation of their retinopathy, which may be due to an outpouring of cortisome during a stress period. Thus the possibility that a hormonal disturbance may be a factor in the causation of the retinopathy cannot be discounted.

It would therefore appear that something more than the effective control of diabetes will be necessary before the elimination of the late manifestations of diabetes becomes possible. Whilst patients are symptomatically better under good control they continue to be dogged by the spectre of the late manifestations.

Good control was maintained with tolbutamide in 42·9% of cases in a middle- to old-aged group at our clinic, and a further 28·3% were kept under fair control.[2] The drug has now been used at the clinic for almost 2 years without any serious toxic effects and it can therefore be considered a valuable addition to our armamentarium in the fight against diabetes mellitus. However, the appearance of cases of retinopathy in the effectively treated group suggests that it will be necessary to continue looking for factors others than hyperglycaemia in its causation and possibly in the occurrence of other late manifestations.

SUMMARY

1. Two cases of diabetes mellitus are described who have developed retinopathy while on tolbutamide therapy.

2. In one case diabetic control was good and in the other it was fair.

3. The question of the occurrence of retinopathy in relationship to the control of diabetes is discussed.

4. The suggestion is made that some other factor besides hyperglycaemia may be the cause of diabetic retinopathy and possibly other late manifestations of diabetes.

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ISLET-CELL ADENOMA OF THE PANCREAS

REPORT OF A CASE

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An adenoma of the islet-cells of the pancreas was first reported by Nicholls in 1902 as a post-mortem finding but no clinical details of the case were recorded. Harris formulated the concept of hyperinsulinism in 1924, 3 years after Banting and Best isolated insulin. In 1927 Wilder and his co-workers were the first to demonstrate an insulin-secreting tumour. The first successful removal of an adenoma was undertaken by Graham in 1929. The diagnostic triad is associated with the names of Whipple and Frantz (1935). This consists of the development of hypoglycaemic symptoms after prolonged fasting or exercise, the dropping of the blood-sugar level below 50 mg.% and the relief of symptoms by the

administration of glucose. Webster[2] has, however, reported a case in which fatal coma ensued with a blood sugar of 64 mg%. Other similar cases have been reported in the literature.

Review of the Literature

A review of the literature on spontaneous hypoglycaemia due to islet-cell tumours of the pancreas shows about 500 reported cases including a case by Lowenthal[1] in South Africa. Extensive reviews of various aspects of the subject are given by Briedahl, Priestley and Rynearson (diagnosis, findings at operation and results),[3] Kelly (diagnosis),[4] Peyster and Gilchrist (medical and surgical treatment),[5] Whipple (pathology, clinical picture, diagnosis, treatment),[6] Howard (surgery),[7] Parfitt (comparison of irreversible coma with that in the insulin treatment of schizophrenic patients),[8] Black *et al.* (review of British cases),[9] Bickerstaff *et al.* (clinical manifestations and diagnosis),[10] Brearley and Laws (carcinoma of the Islets of Langerhans with hypoglycaemia)[11] and Crain and Thorn (medical review).[12]

The interest of the present case lies in the patient's age, epilepsy, family history and associated arteriosclerosis and dementia.

CASE REPORT

History

Mr. H. J. v. H. was first admitted to a mental hospital on 7 June 1946. He was then 68 years old, single, and had been a peasant-farmer all his life. There was a history of *grand mal* epilepsy of 3 years duration. After a fit he became violent and aggressive and this behaviour led to his certification. Neither fits nor abnormal behaviour were observed in hospital and he was discharged on 14 June 1946.

In addition to the above, the patient gave a history of 'minor' attacks of some years duration in which he stated that he felt weak for a few minutes. Apart from a suprapubic scar no physical abnormality was detected in a healthy man. He was described as an abstainer from alcohol.

He was readmitted on 6 December 1946, 6 months later, for similar behaviour, but this was now pre- and post-ictal, the pre-ictal behaviour being described as 'maniacal' by his general practitioner. On admission this time he was grossly confused. During 1947 he had 30 seizures followed by marked restlessness and confusion. Between seizures he was retarded and could give only a poor account of himself. He was, however, quite quiet, cooperative and usefully occupied. During 1948 he had 43 seizures, and apart from this he was confused most of the time. During 1949 the patient had 19 seizures, the confusion was less but his memory for current events was poor, his emotional responses were blunted and his comprehension was impaired. He showed no evidence of an epileptic 'temperament' and was described as being of 'childish behaviour of a good-natured type' and a willing worker.

In 1950 it was noted that fits could be aborted by feeding the patient as soon as his otherwise correct habits became faulty. Apart from the fits the patient had spontaneous attacks of hypoglycaemia and these always responded to glucose. He remained free from seizures until the middle of 1952 and from July 1952 to December 1953 he had 34 seizures. An overt aura of opening and closing his mouth was noticed. He became progressively apathetic and attended only to his own needs. He also became progressively deaf. During 1954, 1955 and 1956 he had 13 seizures and in 1957 he was reported to have bilateral cataracts. His peripheral vessels were arteriosclerotic. His blood pressure was within normal limits. His liver-function tests were found to be normal, his urine was normal and a fasting blood sugar after 16 hours was normal. He was given glucose regularly by day and night and this reduced his hypoglycaemic attacks to 3, but on one occasion intravenous dextrose was required. By the end of 1957 he was deaf, blind, resistive, and interested only in his tobacco. He had to be dressed and his intimate habits were faulty. He spent his day sitting in a chair. On 12 July he went into hypoglycaemic coma and required intravenous glucose to restore him to consciousness. About 12 hours later he began to lapse into stupor and 24 hours later signs of a left hemiparesis were evident. He died, in coma, on 13 July 1958.

Post-mortem examination 36 hours after death showed marked generalized arteriosclerosis. There was a large thrombus in the right lenticulo-striate artery and accompanying cerebral softening. His clinoids were well calcified. There was marked nephrosclerosis and the kidneys were cystic with little normal tissue present. Numerous gallstones were present. In the head of the pancreas a well encapsulated tumour ¾ in. in diameter was found. The pathological report reads: 'The pancreas shows the presence of a "nesidiocytoma" (islet-cell adenoma). This is unfortunately not sufficiently well preserved to demonstrate the cell-type but the clinical history suggests that it must be an insulin-secreting adenoma.'

Family History

1. Brother, W. J. v. H., who was admitted on 24 March 1948 at the age of 64. He was reported to have suffered from epilepsy since 1937 when he was 53 years old. He died 4 months after admission having been confused nearly all the time.

2. Brother, J. M. v. H., who was admitted in 1956 when he was 70 years old. He suffered from senile psychosis and died of bronchopneumonia within 5 months of admission.

DISCUSSION

Family history. Brain[13] states that in a series of 200 epileptic patients there was a family history of this disease in 28%. Lennox, quoted by McV. Hunt,[14] showed that epilepsy occurs among the near relatives 5 times more frequently than in the general population. Kallmann and Sander[15] give the morbidity of epilepsy among siblings as 3·7-3·99% as opposed to an incidence of ·4% and ·28% (in separate studies) in the general population.

If it is accepted that the tendency to seizures may be inherited, and since one sibling of this patient was epileptic, it might be suggested that our patient had a latent epileptic tendency until the necessary trigger, which produced full seizures, became operative. In his particular case there is the hypoglycaemia of an islet-cell adenoma, arteriosclerosis and senility.

With regard to senility, Kallmann[16] gives the expected morbidity rate for the siblings of seniles as 6·5% as opposed to an incidence of 1% in the general population.

Age. This man was 65 years old when his seizures began although the history of attacks of weakness for several years prior to this suggests that he may have been hypoglycaemic then. The age of Parfitt's[8] cases ranged from 30 to 60 years. In an analysis of 20 cases of carcinoma of the islets of Langerhans with hypoglycaemia (Brearley and Laws)[11] the mean age of the patients was 44 years, although there were 3 cases over the age of 65 years. Sixty-five years is also the generally accepted age above which senile psychosis may be diagnosed. The onset of the patient's fits is certainly coincident with the senium. While he showed no evidence of senile dementia on admission, he certainly began to show signs of dementia 3 years later. What part the senile changes *per se* played in his dementia cannot now be determined and the course of a senile psychosis may vary from a few months to years. It will be recalled that his brother, J. M., was admitted at the age of 70 years and died within 5 months.

Epilepsy. The question of the family tendency towards epilepsy has already been discussed. The certifying doctors in December 1946 described maniacal behaviour terminating in a fit and this particular feature in spontaneous hypoglycaemia has been commented on by Briedahl, Priestley and Rynearson.[3] Parfitt[8] gives the incidence of fits in hypogly-

caemia as 50% with a wide variation. He also quotes Crain and Thorn's series in which 28% of 193 cases had fits. On the other hand, it is this type of behaviour, which is not unknown in epileptic psychosis, where the 'mania' can be instantly terminated by an electric convulsive treatment. The incidence of epilepsy in arteriosclerosis is given as 15-20% by Mayer-Gross, Slater and Roth.[17]

Arteriosclerosis. The autopsy showed grossly calcified brittle arteries. A thrombus in one of these was the cause of the patient's death. The extensive renal changes would indicate that the arteriosclerosis had been of many years duration. The incidence of epilepsy in this condition has already been referred to. The rapidity of dementia in this condition is as variable as it is in senile psychosis. Hemiplegia, noted in this patient's terminal phase, occurred in 10% of Crain and Thorn's cases (Parfitt[8]). Black *et al.*[9] reported a case in 1954. Bickerstaff *et al.*[10] suggest that in epilepsy of late onset, in the absence of cerebral tumor, the diagnosis of cortical atrophy or cerebral arteriosclerosis is too easily made and that hyperinsulinism should be excluded in all such cases. Dimsdale[18] analysing 200 cases of epilepsy occurring over the age of 20 found cerebral arteriopathy in 7 cases, all in the over 40-year age group. There were no cases of spontaneous hypoglycaemia in her group.

Dementia. Dementia due to long-standing epilepsy is a daily feature in mental hospitals, where one frequently sees cases with gross dementia who are subject to infrequent fits. The relationship between intellectual deterioration, arteriosclerosis and senility has already been mentioned. Parfitt[8] notes that hypoglycaemia *per se* can produce transient or permanant signs of intellectual deterioration; he gives the incidence as 2%. In Crain and Thorn's series the incidence of irreversible personality changes was about 5% (Parfitt). Webster[2] considers irreversible brain damage among the main dangers of pancreatic adenoma. Black *et al.*[9] report a case with temporary mental abnormality which improved after surgical removal of the tumour.

Nielson and Thompson[19] state that if cortical cells have passed a certain point in sugar deprivation they may require a long time for recovery, even weeks. The episodes of confusion noted in this patient's history may well be evidence of the states implied by Nielsen, but they may also have represented mild hypoglycaemic states or evidence of the signs of temporary intellectual deficit mentioned by Parfitt.[8] However, such episodes are also independently compatible with epilepsy and arteriosclerosis.

SUMMARY

A case of spontaneous hypoglycaemia resulting from an islet-cell adenoma of the pancreas in a man dying at the age of 80 years is reported. The case is discussed from the point of view of his advanced age, epilepsy, family history, arteriosclerosis and dementia.

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HOLIDAY MALADIES AND A HOLIDAY PRACTICE ON THE NATAL COAST

P. LYNCH, M.R.C.S., L.R.C.P., L.M.S.S.A.

A general practitioner in a holiday resort has his own peculiar problems. He is dealing with another doctor's patients to whom he is unknown and the patient-doctor relationship is, in any case, only temporary; therefore he is at a disadvantage from the start. These are additional problems to that of treating the patient's illness.

Each doctor, no doubt, has his own methods. My own experience has taught me not to be too credulous of past histories as related by the patient, and to keep an extremely open mind concerning alleged statements and pronouncements made by the patient's family doctor, since there seems to be a marked tendency among the majority of the holiday patients to be confused about what their doctor has actually meant. Therefore, I always play for safety by sending a note back with the patient outlining their condition and treatment. I have found, too, that in serious and important cases a telephone call or a telegram to the patient's family doctor is extremely helpful and a great pyschological help to the patient and his relatives in establishing a more definite patient-doctor relationship and bringing peace of mind to all concerned.

The illnesses, injuries and problems chiefly met with on holiday in their order of frequency are as follows:

Dermatological Conditions

Sunburn. This is treated in nearly every case by the chemist, and the medical practitioner usually sees only the bad cases and those allergic to the different sunburn lotions. The bad cases of sunburn are best treated on A. P. Codeine, a penicillin injection, phenergan at night (anthisan for the young) and the application of a calamine oily liniment on layers of gauze. It is remarkable how often, through total ignorance of the effects of the sun's rays, the holidays of many people in all walks of life are spoilt.

Solar dermatitis due to different rays of the sun's spectrum is not uncommon. In 17 years of practice I saw none on the highveld, but in the hot humid months at the coast as many as 4 to 6 a week. Sensitivity to the sun is so peculiar that it varies in 50 miles of the same coast in the same person. I have only seen one patient who had also complained of sensitivity 'inland' and he was a Reef practitioner who had received no help from 5 different dermatologists and who was quite yellow from atebrin. Strict protection from the glare and rays of the sun for the rest of the holiday is advised, tannic acid 5% in lannette wax or with 25% alcohol should be applied for protection. Pabavel (paramine benzoic acid) 800 mg. and phenergan ·025 mg. should be given

in daily oral doses. Should the skin be broken, zinc cream with gentian violet, 1 : 5,000 in water, is advisable. In most cases I have advised the patient to have his urine and faeces examined for porphyrins.

Papular urticaria in children is a relatively common condition. In this part of the world, it is due to a sensitivity to bananas in 60% of cases; 5% due to citrus; 5% due to pineapples; 2% due to fish; 2% due to nuts, dates and raisins, and the rest is due to miscellaneous and unknown agents. Impetigo, or the Natal sore or veld sore, often follows due to the scratching. It is more common during a holiday because of the enormous increase in the consumption of the above foods. The banana rash and most of the others respond very well to complete exclusion of the cause and elixir of anthisan syrup; this medication should be given in full doses depending on tolerance in the patient. Simple dietetic measures can be followed even in a hotel, the patients being allowed to eat only what they would eat at home. Calamine with 1% vioform is a good all-round application.

Sandworm disease is a horrible affliction and it is rightly dreaded by all holiday makers who know about it. It is also called creeping eruption. The patient is usually seen after he has been unsuccessfully treated by a chemist, and the infestation is sometimes so multiple that it is serious. Once the exact position of the larvae has been ascertained, the rest is easy. As this can usually be verified by the patient, my procedure is not to charge for the first visit, but to send the patient away with instructions as to how to find the larvae. The tract must be rubbed with cotton wool so that it glows and this procedure must be done 3 or 4 times a day, when the worms' course can be clearly seen. Instructions are also given at the free consultation to take a sedative (sodium amytal barbiturate) some time before treatment, especially in the very young. Provided these instructions have been faithfully followed, freezing with ethyl chloride seldom fails and for good measure I prescribe hetrazan tablets and phenergan for the irritation. Preventative measures against this troublesome and unpleasant disease are to urge the public—and to warn them—always to wear some form of foot-wear on the beaches, and in particular, to avoid sand at the back of the beaches not reached by the sea.

Ear Diseases

Ear diseases are common on a holiday and *otitis externa* is a frequent worry and affects the holiday maker more than the residential population. It occurs mostly in males and is almost certain to occur in all ear scratchers in the hot humid months. The cause of the condition is uncertain and a perusal of a good deal of literature has made it no clearer. Up country mainly the ear scratchers appear to be subject to otitis externa, but at the coast all and sundry are afflicted. An ear itch can be psychosomatic, neurotic or due to various organic dermatological conditions.

The most troublesome form of external otitis is the oedematous desquamatous type, where the discharge is often like chewed-up blotting paper which school boys flick at the ceiling. I wrote to 6 specialists regarding treatment and 6 different types of treatment were suggested. After having carried out these treatments on a trial-and-error basis, I have arrived at the following solution: A warm glycerine and icthyol wick is used in the acute stage. The wick must be packed in tight and used before the oedema occurs making this procedure impossible. A long-acting form of penicillin is injected, sedatives and analgesics are necessary and the application of heat is soothing. On resolution of the oedema I have found that liq. hydrarg. perchlor, glycerine and S.V.R. drops are better than any antibiotic, less expensive, and can be used without seeing the doctor at the slightest symptoms of relapse. Antibiotics have caused extremely severe sensitivity reactions when I have used them.

Sinusitis, despite all the swimming, is not a common disease. *Otitis media* is very common. I do not know whether I am original in my theory, but it is my considered opinion that this sometimes serious and crippling malady, which may result in deafness, is sometimes caused in people, especially children, who are suffering from a heavy cold and travel in fast-moving vehicles from higher altitudes to the coast. I should, therefore, like to suggest that no-one should travel until the acute stage of the cold has passed off, and once over the acute phase, nose drops such as agyrol should be used before and during travelling.

Miscellaneous Diseases

Asthma often spoils a holiday. If I find that a patient remains in a mild state of status asthmaticus I advise him to return home. This is always a disappointment, and often not easy to deal with when the patient has made all arrangements for the holiday. But, since this condition can spoil a holiday, I think it is the most sensible advice I could give. Asthma is more common in a humid sub-tropical climate with a lush vegetation. I know I am going to have asthma cases when there have been showers of rain, a high tide causing a mist from the sea and a hot sultry sun.

Tick-bite is seasonal, but we have cut down the incidence tremendously by removing all cattle from the vicinity of the holiday-makers.

Regarding *honeymoon couples*, it is my practice to have on hand 2 copies of 'Successful Marriage' by Fishbein and Burgess and to lend these to couples afflicted by sexual and other problems. This avoids much time being lost in discussion, and the suggestion that the book is borrowed, read and subsequently returned, is usually accepted with relief. A bride is often subject to cystitis and pyelitis, fortunately usually of a mild type, which quickly responds to appropriate sulfa drugs.

Occasionally a very shy, over-modest, and frightened bride is taken to hospital with a complaint of unsuccessful sexual intercourse or dyspareunia to be examined under an anaesthetic. Examination nearly always shows nothing abnormal and the 'trick' is to pack the vagina with an impressive amount of gauze packing which is only removed when she is again conscious—to reassure both her and the husband together that there is nothing abnormal and that they are two perfectly normal people having almost normal difficulties and troubles.

In *paediatric complaints* infectious diseases in hotels are dreaded both by the parents and hoteliers. One encounters measles, chicken-pox and mumps, but typhoid, paratyphoid, small-pox, meningitis and poliomyelitis are fortunately rare. The common infectious diseases are dreaded because of the close proximity of the children to each other in the hotels, the difficulty of isolation and the high cost of hospitalization in these days. I should like to suggest that all hotels catering for large numbers of children should, in their own interest as well as that of their guests', make provision for a small sick-bay, well away (where this is at all practical) from the rest of the hotel building. In paediatrics mild enteritis is the commonest complaint and it is usually settled by prescribing a suitable proprietary brand of milk such as pellergan and one of the sulfa drugs.

Mild bronchitis is encountered in babies on holiday but, with the exception of children with asthmatical tendencies, they can be easily cured. From my experience it would appear that only the more intelligent mothers take a holiday as they all appear to handle their children expertly without the assistance of the clinics, doctors and welfare visitors, who are to be found around every other block in the bigger centres.

Injuries are very common. Many holiday-makers indulge in all forms of sport: riding, climbing, surfing, and many of them—particularly those who lead sedentary lives—do so with more enthusiasm than caution. Surfing injuries are comparatively rare, but I have had 3 cases of fractured necks because the visitor, through inexperience, attempted to surf in large waves breaking in shallow water.

Foot and leg troubles. Orthopaedic problems are very common, particularly in regard to the lower extremities. One sees a tremendous amount of ruptured plantarus muscles of the calf. Due to the pain, the patient is crippled for 3 or 4 days. The main treatment consists of rest, supportive treatment with an elasto-crepe (and *not* a crepe) bandage, and by raising the height of the heel on the affected side by as much as $\frac{3}{4}$ inch in some cases. The supportive bandage is necessary owing to the bleeding which often accompanies such ruptured muscles. The chief cause of this injury in my opinion, is the change from wearing a shoe with a heel to flat shoes or sandals, and to violent exercise taken on beaches, tennis courts and sports grounds. Foot strain is a common malady among the visitors, and is more marked in the female. A girl with symptomless flat feet at home, who puts on a bit of weight during the year can, as a result, have a miserable holiday. The condition is prevented by using suitable footwear. Fractured toes are common due to the shedding of the normal protective footwear.

Drowning is, fortunately, a rare occurrence, and is likely to

be even rarer now we have the shark-protection nets. It may be of interest to record that I attended 4 fatal shark injuries in the last year, and the most startling thing was the completely ensanguinated appearance of the corpses. It was not established whether this was caused by shock or by bleeding from the large wounds.

Psychological disturbances are common and it is surprising how many men and women become mentally upset whilst on holiday. They visit one in an abject state of mind, obviously disturbed by leaving their work and having nothing to occupy their minds, and suicide, although not common, has been known to occur.

In the very emotionally upset and depressed patient, it should be borne in mind by the family practitioner that a holiday should only come *after* psychiatric advice. It is my practice to see such cases twice a week, give them some supportive advice, prescribe sedation for them during the day and see that they sleep soundly at night, and to encourage them to find something with which to occupy themselves. This is often difficult, for the majority of cases are not interested in the usual trivial holiday pursuits.

From my observations, I have come to the conclusion that many of our top executives, and men and women holding important government and business positions, are taxing themselves beyond their capacities, and this strain is very apparent when they visit a doctor whilst on holiday. In many cases the job is too big for the man: his promotion has been too rapid and his background too poor to cope with the demands of an expanding economy.

A simpler psychological holiday complaint, yet one which is very difficult to handle, is that of the child (up to 5 years old) who has not yet become adjusted to the so-called oedipus complex, and who, instead of seeing his father for perhaps one hour every day at home, now sees him all day on holiday. These children sometimes behave so badly and bewilder their parents so completely that they spoil the holiday for the whole family. For those who may find this difficult to believe, Benjamin Spock's sensible book provides the complete answer.

The alcoholic is seldom encountered in a holiday practice. The governing factor here would appear to be one of finance, since the alcoholic is seldom in a position to be able to afford a holiday. When alcoholics are seen, they are encouraged to return home and join the A.A. The A.A. seems to have special methods of taking care of their 'arrested' members on holiday.

We also encounter the insecure, dissatisfied, and the really sick patient who wants to check up on his own doctor whilst away from home. This is more marked in those who come from areas where it is difficult for the patients to obtain a second opinion. A blend of discretion, tact and honesty is required, especially if one comes across the undiagnosed diabetic or the grumbling Medical Benefit Society patient with a paranoid personality.

Malaria is of course the commonest tropical disease seen, and is often precipitated by swimming in a cold current or on a cold day.

When death occurs on holiday it seems even sadder than at any other time, and it is particularly distressing for the bereaved relatives, who may be many hundreds of miles from home, or who may have to travel many miles from home to make the necessary arrangements.

The Provincial Hospital service causes concern because the hospital really only caters for the residential population. With the influx of visitors the bed problem becomes acute. It is sometimes the practice to admit no 'cold' cases during the holiday season. Tonsillectomy for the local children then becomes quite a problem.

Book-keeping in a holiday practice presents its own problems. A practice changes every month and the doctor's memory is often unmercifully taxed. Matters are not made easier by the numerous medical aid society forms, and I cannot help feeling that the medical practitioner is exploited by these societies, for, in addition to rendering services at reduced fees, he is also compelled to do the Societies' book-keeping gratis.

Seldom, if ever, is a practitioner saddled with a bad debt from a holiday patient, and accounts are always settled promptly, usually before the patient leaves for home. Apparently the financially insecure cannot take a holiday.

In conclusion, may I say that a holiday practice, despite its difficulties, has its compensations and rewards, and offers challenges that are perhaps not found in other types of practice. One is judged not only by one's patients but by the patients' doctors. In turn, there is the opportunity of judging the work of one's colleagues all over the continent, and this aspect is often most revealing.

SUMMARY

The commonest holiday maladies and complaints such as dermatitis, otitis externa, otitis media due to travelling from a high to a low altitude, and special psychological states are mentioned and their treatment suggested. Problems in general practice at the coast are mentioned and advice tendered.

SYMPOSIUM ON EXTRA-CORPOREAL CIRCULATION

A symposium on extra-corporeal circulation will take place on Saturday 31 January and Sunday 1 February 1959 at the Medical School, Hospital Hill, Johannesburg, under the auspices of the Faculty of Thoracic Surgery of the College of Physicians, Surgeons and Gynaecologists of South Africa. The meetings will be open to all interested, and further particulars may be obtained from the Hon. Secretary, Mr. D. N. Fuller, Clarendon Centre, Parktown, Johannesburg.

Saturday 31 *January*

9.00-9.20 a.m. Pharmacology of the heart: Dr. P. A. Bradlow.
9.20-9.40 a.m. Metabolism of the heart: Dr. M. Zion.
10.30-11.30 a.m. Some problems of pump oxygenation: Prof. D. Rosenberg.
11.30 a.m.-12.30 p.m. Development of heart and great vessels: Prof. P. Tobias.
Development of septal defects: Mr. L. du Plessis.

2.15-3.00 p.m. Demonstration of pump oxygenators (at Brenthurst Clinic): Mr. D. N. Fuller.
3.15-4.15 p.m. Results of experimental work: Mr. C. Barnard, Mr. P. Marchand and Mr. J. C. van der Spuy.
4.30-5.15 p.m. Open panel discussion.

8.00-9.00 p.m. Indications for operation: Dr. L. Braudo and Dr. L. Vogelpoel.
9.00-10.00 p.m. Results in clinical cases: Mr. W. Phillips and Mr. D. Adler.

Sunday 1 *February*

9.00-10.45 a.m. Problems of pump oxygenation.
(*a*) Blood collection and storage: Dr. M. Shapiro.
(*b*) Anaesthesia: Dr. B. Meaker.
(*c*) E.E.G. monitoring: Dr. Berman.
(*d*) Blood replacement: Dr. W. Scott.
(*e*) Pressures and E.C.G.: Dr. M. Zion.
(*f*) Biochemical and haematological control: Dr. H. B. Greig.

11.15 a.m.-12.45 p.m. Post-operative care:
(*a*) Surgical: Mr. D. Adler.
(*b*) Haematological and biochemical: Dr. H. B. Greig.
(*c*) Medical: Pacemaker and monitor: Dr. Klugman.
Cardiac: Dr. L. Vogelpoel.
Fluid balance: Dr. W. Scott.

2.00-4.00 p.m. Surgical techniques: Mr. C. Barnard and Mr. D. Fuller.
Management of pump oxygenators: Mr. C. Barnard and Mr. L. du Plessis.

The following have been invited to preside at the various sessions: Prof. D. J. du Plessis, Prof. J. Gear, Prof. H. W. Snyman, Mr. L. Fatti, Dr. M. Nellen, Dr. M. M. Suzman, Mr. W Trubshaw, Dr. B. van Lingen, Dr. V. Wilson.

42nd MEDICAL CONGRESS, EAST LONDON : 42ste MEDIESE KONGRES, OOS-LONDEN

The organizing committee of the 42nd Medical Congress (M.A.S.A.) announce that Prof. George Burch, Cardiologist, of Tulane University, New Orleans, USA, and Sir Russell Brock of Guy's Hospital, London, have accepted invitations to attend Congress.

Die Organiserende komitee van die 42ste Mediese Kongres (M.V.S.A.) kondig aan dat prof. George Burch, Kardioloog, van die Universiteit van Tulane, New Orleans, VSA, en Sir Russell Brock van Guy's Hospitaal, Londen, die uitnodigings aangeneem het om die Kongres by te woon.

THE COLLEGE OF PHYSICIANS, SURGEONS AND GYNAECOLOGISTS OF SOUTH AFRICA : DIE KOLLEGE VAN INTERNISTE, CHIRURGE EN GINEKOLOË VAN SUID-AFRIKA

EXAMINATIONS

The forthcoming examinations of the College will take place at the University of Cape Town on 4 May 1959 and the *viva* examinations on 7 and 8 May 1959:

1. The Primary Examinations for the Fellowship of the College of Surgeons.
2. The Final Examination for the Fellowship of the College of Surgeons.
3. The Fellowship of the College of Physicians.
4. The Fellowship of the College of Obstetricians and Gynaecologists.
5. The Diploma in Midwifery.

Full details and forms of enrolment may be obtained from the Hon. Registrar of the College of Physicians, Surgeons and Gynaecologists of South Africa, P.O. Box 120, Cape Town.

T. B. McMurray
Honorary Registrar

EKSAMENS

Die aanstaande eksamens van die Kollege sal op 4 Mei 1959 by die Universiteit van Kaapstad afgeneem word en die *viva* eksamens op 7 en 8 Mei 1959:

1. Die Primêre Eksamen vir die Lidmaatskap van die Kollege van Chirurge.
2. Die Finale Eksamen vir die Lidmaatskap van die Kollege van Chirurge.
3. Die Lidmaatskap van die Kollege van Interniste.
4. Die Lidmaatskap van die Kollege van Obstetrici en Ginekoloë.
5. Die Diploma in Verloskunde.

Verdere inligting en aansoekvorms is van die Ere-Registrateur van die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika, Posbus 120, Kaapstad, verkrygbaar.

T. B. McMurray
Ere-Registrateur

EIGHTEENTH CONGRESS OF THE INTERNATIONAL SOCIETY OF SURGERY, MUNICH, 13—20 SEPTEMBER 1959

The major subject for discussion at this Congress will be *Grafts* and it has been decided to deal with the subject under the following headings: (1) The biology of grafts (summary of the *colloque* held at Liege in March 1959), (2) skin grafts, (3) bone grafts, (4) organ grafts, and (5) arterial grafts.

Members of the Society who wish to take part in the discussions are asked to inform the Secretary of their intention to do so by 15 July 1959 at the latest and those who wish to read short papers on clinical subjects must inform the General Secretary by 15 May 1959. A Forum during the meeting will be open to non-members (if approved by the Scientific Council) and 2 copies of their text and a summary of not more than 10 lines must reach the Secretary by 15 June 1959. Further information may be obtained from the Secretary General, Prof. P. Martin, 141 rue Belliard, Bruxelles, Belgium.

The provisional programme of the Congress is as follows:

Monday 14 *September*.	Morning—reports and discussions on grafts Afternoon—reports and discussions on grafts
Tuesday 15 *September*.	Morning—forum Afternoon—forum
Wednesday 16 *September*.	Morning—individual papers Afternoon—individual papers
Thursday 17 *September*.	Morning—forum or individual papers Afternoon—general assembly
Friday 18 *September*.	Morning—forum held at a joint meeting with the International Society of Cardiovascular Surgery Afternoon—individual papers
Saturday 19 *September*.	Morning—arterial grafts.

UNIVERSITY NEWS : UNIVERSITEITSNUUS

Prof. J. H. Louw, Professor of Surgery at the University of Cape Town, has left South Africa on an overseas travel and study trip. This trip has been made possible by a Carnegie Corporation Grant for travel and study in the USA and Professor Louw has also had additional assistance from Pfizer Laboratories (South Africa) (Pty.) Ltd.

While he is in America Professor Louw will visit surgical centres in Boston, New York, Baltimore, Philadelphia, Washington, Houston, Chicago, Minneapolis, Rochester and Iowa and since his main interests are in paediatric and vascular surgery he hopes to meet leading American doctors of these fields.

Professor Louw will also give a series of lectures at the University of Minnesota, the Iowa State University, the Mayo Clinic, and in New York and other centres. Among the subjects of these lectures are: Carcinoma of the Thoracic Oesophagus with Special Reference to the Disease in the South African Bantu; Congenital Intestinal Atresia—Observations on its Pathogenesis and Treatment; Intestinal Malrotation and Duodenal Ileus; Some Aspects of Hirschsprung's Disease with Special Reference to this Disease in the Newborn; Large Abdominal Lumps in Children with Particular Reference to Embryoma of the Kidney; and Anorectal Malformations.

After 3 months in America Professor Louw will travel to the UK as he has been granted the Moynihan Lectureship at the Royal College of Surgeons of England. This lecture will take place on 24 April and the subject will be 'Intestinal Atresia'. This is the first time that a South African has been given the honour of a Moynihan Lectureship. The Moynihan Lecture forms part of the programme of the Congress of the Association of Surgeons of Great Britain and Ireland. Professor Louw will possibly also lecture in Edinburgh on 'Carcinoma of the Oesophagus'. He has also been invited to give lectures at Guy's Hospital, the Postgraduate Medical School, the University of Liverpool and the Royal College of Surgeons.

On his return trip to South Africa Professor Louw hopes to spend some time in Rome, where Professor Pietro Valdoni, Professor of Pathological Surgery at the University of Rome, has invited him to lecture.

UNIVERSITY OF NATAL: EXAMINATION RESULTS, DECEMBER 1958

The following candidates have completed the requirements for the M.B., Ch.B. degree:

Atcha, E. I.
Bhagwandeen, S. B.
Makgalemele, V. R.
Mdlalose, F. T.
Moreosele, A. T.
Pather, M. R. (Miss)
Piliso, T. L. (Miss)
Seedat, I. M.
Vaid, A. A. (Miss)

The following prizes have been awarded to the graduands in question:

Association of Surgeons Prize: Dr. S. B. Bhagwandeen
Burroughs Wellcome Prize: Dr. S. B. Bhagwandeen
Arthur Goldsmith Prize: Dr. V. R. Makgalemele
Prize in Medicine: Drs. S. B. Bhagwandeen and I. M. Seedat
Prize in Paediatrics: Dr. E. I. Atcha.

J. S. DU TOIT-GEDENKFONDS : J. S. DU TOIT MEMORIAL FUND

	£	s.	d.
Previously acknowledged: Voorheen erken	95	12	6
Dr. Alex. Smith	2	2	6
Dr. F. Schneider	5	5	0
Drs. W. H. and W. E. Lawrence	5	5	0
Nasionale Pers Beperk	25	0	0
Drs. H. Penn and A. E. Amoils	5	5	0
Dr. J. P. de Villiers	5	5	0
Dr. M. Helman	5	5	0
Dr. F. Krone	10	10	0
Dr. H. C. Wykerd	5	5	0
Dr. C. J. B. Muller	10	10	0
Totaal soos op 31 Desember 1958	£175	5	0
Total as at 31 December 1958.			

HONORARY TREASURER'S SPECIAL FUND : ERE-PENNINGMEESTER SE SPESIALE FONDS

The following donations during the months of November and December 1958 are gratefully acknowledged:
Met hartlike dank word die volgende skenkings gedurende November en Desember 1958 erken:

	£	s.	d.
Previously acknowledged: Voorheen erken ..	1,129	3	0
Dr. I. A. van Niekerk, Bellville	5	5	0
Dr. A. G. Park-Ross, Kroonstad	4	4	0
Dr. J. Black, Johannesburg	5	5	0
Dr. W. J. Naude, Franklin, K.P.	7	7	0
Dr. H. Grant-Whyte, Durban	10	10	0
Dr. H. S. Gear, W.H.O., Geneva	2	0	0
Total as at 31 December 1958	£1,163	14	0
Totaal soos op 31 Desember 1958.			

PASSING EVENTS : IN DIE VERBYGAAN

Dr. S. I. Mendel, M.B., B.Ch. (Rand), has changed his address to 82 Medical Centre, Field Street, Durban, as from 1 January 1959. Telephones: Rooms 22419, 20801, residence 58648, after hours and emergency 29326.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria. Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

* * *

Correction. In the report on *The Treatment of the Nephrotic Syndrome with Particular Reference to the Steroids and the Diuretics* by Professor Eales and Dr. Saunders, which appeared on page 1222 of the *Journal* of 20 December 1958, a correction is to be made in the 4th sentence of the 2nd paragraph. This should read 'Treatment included the following: ACTH 52 courses, cortisone 10 courses, prednisone 35 courses, prednisolone 7 courses, triamcinolone 2 courses, and dexamethazone 5 courses'.

* * *

Dr. André Beyers, M.B., Ch.B. (Cape Town), M.D. (Clin.) (Pretoria), M.Med. (Rad.) (Pretoria), has joined Drs. Muller and Keet, Radiologists, at 206 Dumbarton House, corner of Adderley and Church Streets, Cape Town, as from 1 January 1959

Dr. André Beyers, M.B., Ch.B. (Kaapstad), M.D. (Klin.) (Pretoria), M.Med. (Rad.) (Pretoria), het vanaf 1 Januarie 1959 as 'n radioloog aangesluit in praktyk met drs. Muller en Keet te Dumbartongebou 206, hoek van Adderley- en Kerkstraat, Kaapstad.

* * *

South African Journal of Laboratory and Clinical Medicine. Members are reminded that subscriptions to the quarterly *Journal* of the Association are £1 5s. 0d. per annum. This *Journal* comprises articles of a scientific and technical nature which do not usually appear in the weekly *Journal*, but are of interest to all members of the medical profession.

Suid-Afrikaanse Tydskrif vir Laboratorium- en Kliniekwerk. Lede word daaraan herinner dat die inskrywingsgelde vir die kwartaalblad van die Vereniging £1 5s. 0d. per jaar is. Hierdie *Tydskrif* bevat artikels van 'n wetenskaplike en tegniese aard wat nie gewoonlik in die weeklikse *Tydskrif* verskyn nie, maar wat van belang vir alle lede van die mediese professie is.

* * *

The Radiological Society of South Africa (Transvaal Branch) wishes to draw attention to a lecture to be given by Prof. A. S. Johnstone of Leeds, England, on Wednesday 28 January 1959, at 8.15 p.m. The venue will be Medical House, Esselen Street, Johannesburg. The subject is 'Recent observations on the radiology of the gastro-oesophageal junction'. Professor Johnstone was formerly professor of diagnostic radiology at the University of Cape Town and now holds the Chair at Leeds. He is a recognized authority on this subject and was a guest of the Radiological Society of North America at their meeting in November 1958, when he spoke on this subject. Apart from radiologists, all those concerned with the diagnosis and management of diseases of the gastro-oesophageal junction are urged to attend.

* * *

The South African Medical and Dental Council. At the first meeting of the new Council Prof. S. F. Oosthuizen, of the University of Pretoria and Head of the Radiological Department of the Pretoria Hospital, was re-elected President. Professor Oosthuizen was President during the 5 years of the last Council and for about 3 years of the previous Council. Prof. H. W. Snyman, Professor of Medicine and Dean of the Faculty of Medicine, Pretoria University, was elected Vice-President.

The following members of the Council were elected to the executive committee: Prof. S. F. Oosthuizen, Prof. H. W. Snyman, Dr. J. J. du Pré le Roux, Dr. R. Lance Impey, Dr. P. F. H. Wagner, Prof. E. H. Cluver and Prof. J. Black.

* * *

Wyle prof. M. C. Botha. Met die heengaan van prof. M. C. Botha op 3 Januarie 1959, het die mediese professie 'n goeie vriend en

groot voorvegter verloor. Wyle professor Botha se veelvuldige prestasies is goed aan ons almal bekend. Hy was Hoof van die Departement Afrikaans-Nederlands aan die Universiteit van Kaapstad, Superintendent-Generaal van Onderwys in die Kaapprovinsie, Sekretaris van Unie-Onderwys en Rektor van die Universiteit van Pretoria. Dit is veral in sy hoedanigheid as Rektor van die Universiteit van Pretoria dat sy belangstelling in die mediese professie aan die lig gekom het. Een van die groot take van sy lewe was om die grondslae te lê vir die oprigting en uitbouing van die Mediese Skool van die Universiteit van Pretoria en daarmee saam die skepping en ontwikkeling van geneeskundige terme in Afrikaans. Die voorstel om 'n Afrikaanse mediese skool te Pretoria te stig, sowel as die voorstel om 'n mediese skool vir Nie-blanke studente te stig het albei oorspronklik van professor Botha gekom. As geneeshere is ons almal baie dank aan hom verskuldig en ons wil ons waardering graag hiermee in alle opregtheid betuig.

* * *

The Southern African Cardiac Society. A meeting of the Cape Province Section of this Society was held at Groote Schuur Hospital, Observatory, Cape, on 18 December 1958, at 8.15 p.m. The programme was as follows:

1. Dr. C. N. Barnard discussed the surgical anatomy of the tetralogy of Fallot and then showed a ciné film of an operation for total repair of tetralogy of Fallot performed at the Lillehei Cardiac Surgical Centre, University of Minnesota, Minneapolis, USA.

2. Dr. M. Nellen described a case of congenital heart disease in a girl aged 12. This patient had severe congenital aortic stenosis, severe congenital pulmonary stenosis, atrial septal defect and partial aberrant pulmonary venous return. The diagnosis was proved by cardiac catheterization. It was stressed that the combination of pulmonary stenosis and aortic stenosis, although reported before, is extremely rare. The patient is very shortly due for an attempt at complete repair of these defects under the by-pass heart-lung machine. Aortic valvotomy must precede pulmonary valvotomy.

3. Dr. L. Vogelpoel described the effect of amyl nitrate inhalation on the ejection systolic murmurs in the above case. The slight and delayed increase in loudness of the pulmonary systolic murmur was unusual for pulmonary stenosis alone and was explained on the basis of the presence of severe aortic stenosis preventing the systemic 'tidal wave' effect occurring, thus vitiating the usual increased volume return to the right heart.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

SILM STAIN REMOVER

Maybaker (S.A.) (Pty.) Ltd., announce the introduction of 'Silm' brand fixer-stain remover. Staining is a short-coming of all liquid ultra-rapid fixers and 'Silm' has been formulated so that it is suitable for eradicating photographic silver stains from all kinds of fabric, white or coloured, coarse or delicate. 'Silm' is sprayed over the stained area and the marks will normally disappear within minutes of this operation. After treatment a liberal application of water is required. Where stain is extensive, it is advisable to have the garment laundered after treatment. 'Silm' is likely to be of great interest to all users of photographic chemicals for the protection and care of clothing such as white overalls.

NIVAQUINE

Maybaker (S.A.) (Pty.) Ltd., announce a new presentation of 'Nivaquine' brand chloroquine sulphate in the form of 68 mg. (equivalent to 50 mg. base) tablets for use as a daily suppressive dose in malaria. In West Africa, where malaria is hyperendemic, experience has shown that if suppressive doses of antimalarials are not taken daily any other dosage schedule is likely to be overlooked. The existing tablet, containing the equivalent of 150 mg. chloroquine base, is more than is necessary for daily suppressive dosage; moreover, there is the cost factor of the tablet of this strength and the possible increase of disagreeable side-effects. After field trials in Africa it was decided that a tablet containing the equivalent of 50 mg. chloroquine base would be acceptable as the standard daily suppressant dose. In addition, the new 50 mg. base tablet should be useful for therapeutic use in children by overcoming the need to employ divided 150 mg. base tablets. Besides malaria, 'Nivaquine' has been used successfully in extra-intestinal amoebiasis, in lupus erythematosus, in certain vesicular dermatoses, in photosensitivity reactions such as those caused by drugs, in dermatitis, lichen planus, in the elimination of tapeworm, and in the long-term therapy of rheumatoid arthritis.

ELECTRON MICROSCOPE FOR CANCER RESEARCH

Metropolitan-Vickers South Africa (Pty.) Ltd., introduce the latest model Metropolitan-Vickers Electron Microscope, and supply the following information:

The Royal College of Surgeons of England has recently installed a Metropolitan-Vickers Electron Microscope, Type EM6. This equipment has been purchased for the College from funds provided by the British Empire Cancer Campaign, and will be used for research work with particular reference to cancer. This British instrument embodies the most notable advances in electron optical design, together with a variety of features contributing to results of the highest quality. It has a magnification of 120,000X, and for critical examination of the image incorporates a binocular telescope with a further magnification of 10X. The resolving power is 15 Angstrom units.

Two earlier models of electron microscopes made by this Company have been in use most successfully in this country for a number of years. One is installed at the Poliomyelitis Research Institute, Johannesburg, and the other is installed at the Chamber of Mines Research Laboratory, Johannesburg, where it is employed on dust-particle research. A third instrument will be installed early this year at the University of Cape Town, where it will be used in research in the medical, metallurgical and physical science fields.

Full particulars of these instruments can be obtained from Metropolitan-Vickers South Africa (Pty.) Limited, P.O. Box 3633, Johannesburg.

CORRESPONDENCE : BRIEWERUBRIEK

A PSYCHIATRIC APPROACH TO INFERTILITY

To the Editor: Female infertility, looked upon from a psychiatric point of view, is a subject of considerable interest to me. The appearance of an article by Murless[1] on tubo-uterine spasm in a recent issue of the *Journal*, makes this an opportune moment to express my views.

I am sure that general practitioners and gynaecologists will agree that in a high percentage of cases which they investigate, they are unable to establish an organic basis for infertility. Like Murless, I am convinced that the infertility in these cases is due to uterine irritability and tubo-uterine spasm of functional origin. Murless is undecided on whether an inherently defective autonomic nervous system or mental conflict should be held culpable

for the spasm which occurs. I would like to suggest that it is a state of emotional tension brought to bear on an inherently predisposed autonomic nervous system that results in tubal spasm, functional occlusion of the tubes and consequent infertility.

With very obvious trepidation, Murless suggests the possibility of the use of tranquillizing drugs in the treatment of these cases of infertility. Surely, once organic causes have been excluded, infertility must be accepted without reserve or hesitation as a purely psychogenic problem. I am satisfied that the only rational approach to the treatment of these cases lies in treating the underlying emotional tension. The suggestion that tranquillizing drugs should be used is therefore theoretically sound, but practical experience with these drugs has been such as to suggest that this is not likely to solve the problem.

Up to the present time, the possible value of psychotherapy in the treatment of infertility has not received the consideration it deserves. While I am well aware of the criticism to which I might be subjected, I have no hesitation in expressing the view that psychotherapy should be the method of choice in the treatment of a case of female infertility where organic pathology has been excluded. Furthermore, provided a young married woman is cooperative, of average intelligence and endowed with a reasonably well integrated personality, psychotherapy should offer her a very reasonable chance of overcoming infertility.

I am suggesting, then, that female infertility is very often a psychogenic disorder, that it is best treated through the medium of psychotherapy and that, in well chosen cases, this form of treatment should yield favourable results.

817 Medical Centre Harold Cooper
Cape Town
29 December 1958

1. Murless, B. C. (1958): S. Afr. Med. J., 32, 1209.

REPORTS ON MEDICAL EXAMINATIONS

To the Editor: Quite frequently insurance representatives ask me to hand completed medical forms to them rather than post them to head offices, although it is stated on the forms that they should be sent to the head office only. In some cases I am given envelopes which are addressed to a branch manager.

If this procedure is incorrect, I am of the opinion that insurance companies should be instructed to inform their agents that they should not ask medical examiners to hand completed medical forms to them. Could a statement be published in your *Journal* without publishing my name?

27 December 1958 *General Practitioner*

[Reports on medical examinations for insurance purposes are confidential documents. By refusing to hand completed reports to persons other than those indicated on the forms, medical practitioners are complying with the ethical rule on professional secrecy of the South African Medical and Dental Council.—*Editor.*]

DIE MEDIESE RAAD

Aan die Redakteur: Vergun my die geleentheid om deur middel van die *Suid-Afrikaanse Tydskrif vir Geneeskunde* die tien gekose lede van die Mediese en Tandheelkundige Raad, geluk te wens met hulle verkiesing en hulle 'n vrugbare tydperk van diens in belang van geneeskunde toe te wens.

G. F. C. Troskie

Döngesstraat 18
Kroonstad
5 Januarie 1959

CRITICISMS OF THE JOURNAL

To the Editor: May I as a subscriber of many years standing and an occasional contributor to the *South African Medical Journal* congratulate you on your appointment to the important and responsible position of Editor, and express the hope that you will successfully guide its destinies for many years to come. It would however be an ostrich-like attitude to pretend that there is no room for improvement in our *Journal* and to fail to face up to its shortcomings. I, therefore, trust you will receive these comments in the spirit in which they are offered.

In recent times a number of rival journals have sprung up in the North, which seriously threaten the supremacy of our *Journal*. Among the medical profession there is grave dissatisfaction with the *Journal* and many only continue to receive it because it comes as part of their Association membership. These facts demand urgent consideration of the *Journal's* defects and an attempt to remedy them.

First of all I would mention the low scientific standard of too many of its articles. Contributions are accepted for publication which simply have no right to appear in a journal of its standing. And it is no use answering that these are the best on offer—a higher standard must be cultivated among our medical authors. Encouraging signs of this have recently been noticeable in some contributions from our medical schools.

Among the type of article I would beg you adamantly to refuse is the one typified by 'A Case of Von Piffelheim's Disease (non-ossifying peri-urban dyslexia)', wherein the author adds yet one more to the seven already described by the learned professor in the *Deutsche Algemeine Paperospapier* for 1906. Such a paper is completely unimportant to your readers (or even to the late lamented Professor Von Piffelheim). It merely serves as a means of self-advertisement for a lazy medico ambitious to see his name in print.

The essential test of the suitability of a contribution for publication should be whether it adds anything to the sum total of medical knowledge. If it does so, to however modest a degree, it merits consideration. But there must be a conscious effort by the author to demonstrate something by scientific investigation. And you cannot do this merely by quoting from other people's writings (except perhaps in the case of the history of medicine). Thus a careful description of the effect of a drug on even one or two patients is scientific investigation, while the addition of yet another case to a previously described, rare and obscure, pathological condition, is mere self-advertisement.

There is a widespread demand among general practitioners for the didactic type of article wherein a man of eminence in a particular branch of the profession, tells how to do certain things. Features of this type, contributed by invitation, should appear frequently, but should come only from those at the very pinnacle of the profession.

While a good deal of dull 'medical politics', such as is relegated to the supplement in the *British Medical Journal*, has to appear in our *Journal*, yet when an important matter like the threat to the dispensing rights of doctors was in the air, our *Journal* appeared to treat the matter with almost Olympian detachment. In fact it permitted a rival, privately issued journal, to take the initiative in rousing us to a sense of danger, whereas it should have stood in the van of the campaign against the threat to our profession from the very beginning.

Turning now to the other extreme, a weakness of our *Journal* is its poverty in items of what may be called 'social and personal' news about the profession. In a country of long distances it is important to keep old friends in touch with each other's doings. I am aware of the attempts previously made to remedy this deficiency and of the obstinate failure of the profession to cooperate in supplying the necessary news items. May I suggest that you should turn to another source. Each branch of the Association has a secretarial assistant, sometimes only part-time. Offer this lady 2s. 6d. for each personal news item submitted and published, and I am sure you will get all you need. She is in an ideal strategic position to collect such items, and her facts can always be checked by the branch secretary.

These suggestions are made in no spirit of captious criticism, but with a sincere desire to see our *Journal* become worthy of its status, for by it we are judged all over the world. Even if this letter merely provokes discussion it will have achieved its aim.

2 January 1959 M.M.

[The type of article published still depends mainly on what is available. There has been a general improvement in articles submitted and when more are obtained of the kind our correspondent likes, fewer will appear of the sort he deplores. Certainly it is not because better articles are rejected that some of a less important nature are published. The Association's *Journal*, however, ought always to afford a medium for members trying their prentice hand at medical writing—reports of interesting conditions encountered, for example. As to articles of a didactic nature, we have published more than one series of these and this year have started another series. With regard to the attack on the right of doctors to dispense, the *Journal* by no means remained silent during this crisis; we may refer to editorials on the subject published on 9 June and 11 August 1956 (30, 534 and 753) and other articles that appeared. We welcome suggestions for the improvement of the *Journal*, but we have good reason to believe that it is not held in such low esteem by informed opinion generally as our correspondent would suggest.—*Editor.*]